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DIVERGENCE EXCESS

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At the Tenth International Congress of Ophthalmology in 1904, A. Duane read a paper on the evolution of squint. In his opinion nonparalytic divergent squint develops either from insufficiency of convergence or from excess of divergence or from a combination of the two. He considered divergence excess to be produced by overactivity of the center controlling the divergence movements. In his opinion it is secondary in about two thirds of the cases, following primary insufficiency of convergence, and is primary in only one third, manifesting itself in the beginning as marked exophoria with excessive prism divergence for distance vision, but only slight exophoria for near vision and with a continuing normal near point of convergence. Gradually there develops manifest squint for distance with a further increase in the prism divergence, but the near point of convergence is still normal. Finally, after a gradual recession of the near point, the angle of strabismus is the same for distance and near vision.

Dunnington [1] fully agreed with Duane in regard to the etiology of divergent strabismus, considering it as primary or secondary divergence excess due to overstimulation of the center of divergence. In Dunnington's opinion this overstimulation in primary divergence excess is caused by a congenital lesion, whereas in secondary divergence excess it is the result of an untreated severe convergence insufficiency. I have long been skeptical of the correctness of this theory. I cannot believe that all, or even the majority, of the cases of nonparalytic divergent squint are based on overactivity or overstimulation of the divergence center. My doubts are based on both theoretical considerations and clinical experiences. There are strong reasons for the assumption of a divergence innervation antagonistic to the convergence innervation. And the characteristic symptoms of divergence paralysis must also, in my opinion, be caused by a lesion of the divergence center or of the pathway descending from the sixth nucleus. But the diagnosis of divergence excess does not seem to me to have been sufficiently accounted for by

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1. Dunnington, J. H.: Divergence Excess: Its Diagnosis and Treatment, Am. J. Ophth. **56**:344, 1927.

Duane and Dunnington. They do not take into consideration one of the most important factors in the etiology of divergent strabismus, namely, the anomalous position of rest dependent on purely mechanical conditions, which vary within wide limits in different cases. When through illness or trauma one eye becomes blind or amblyopic or aphakic, so that the fusion tendency is lost, in the majority of cases there is gradual development of squint. Divergence of more than 2 degrees develops in from 60 to 66 per cent of cases, and less than 2 degrees in from 10 to 30 per cent. The divergence is combined with a vertical deviation in about 10 per cent, whereas convergent strabismus arises in 10 per cent of adults and in 16.6 per cent of children under 15 years of age. These differences cannot be explained as due to different intervals of time between the loss of the fusion tendency and the time of investigation, since more than 60 per cent of my extensive series of patients had lost binocular vision more than four years before the investigation was made. That in consequence of the loss of fusion tendency such different kinds of squint occur and that in some cases there is no squint must depend mainly on the different position of rest in individual cases. Moreover, all ophthalmologists can recall cases of convergence paralysis or marked convergence insufficiency with orthophoria or even esophoria for distance, whereas other cases of the same anomaly present exophoria or divergent squint. Here, too, the difference is based on the variation in the primary position of rest in individual cases. Such developments are not reconcilable with Duane's assertion that the continuous inactivity of the center for convergence movements leads to persistent overactivity of the center for divergence movements, resulting after a while in divergence excess. Duane referred for support of his hypothesis to Sherrington's law of reciprocal innervation, according to which the stimulation of an oculomotor center in the cortex causes at the same time the contraction of the agonist muscles and the relaxation of the antagonists. But I do not think it possible or justifiable to infer from this law that continuous inactivity of the center of convergence leads to overstimulation of the center for divergent movements. In addition to these theoretical reasons many clinical experiences contradict Duane's conclusion.

However, though declining the theory of divergence excess as the general cause of divergent squint in the sense of Duane, I must acknowledge the occurrence of divergence excess as a definite anomaly with characteristic signs and symptoms. The following case forced me to make a diagnosis of divergence excess as the foundation of the trouble.

REPORT OF A CASE

After an attack of influenza, in 1918, a woman 22 years of age noticed impairment of vision. The first examination of the eyes revealed nothing abnormal. A year later the ophthalmologist found a spasm of accommodation. Full vision could

be obtained in the beginning with a 1 diopter concave lens; after a week, 4 diopters were required, and a few days later, 7 diopters. Yet a year before the patient had had full vision with a convex glass of 1 diopter. She was given treatment with atropine; after a few weeks she noticed double vision. Prism spectacles caused discomfort. Hypnotic treatment and a long stay in a sanatorium proved vain.

In 1922 the patient was sent to me. On testing the abduction power I obtained with prisms of 14 degrees, base in, put before both eyes, the complete relaxation of the fusion convergence, by which exophoria had been kept latent. Afterward without prisms the exophoria was 12 degrees. The spasm of accommodation was due to the convergence impulse which was necessary to overcome the exophoria. Before her bodily strength had been weakened by influenza the patient had been able to overcome the exophoria without any trouble, that is, without producing a spasm of accommodation. After the illness the relative range of convergence was restricted so that the connection between convergence and accommodation could not be loosened sufficiently, and this manifested itself in the spasm of accommodation which accompanied the effort to overcome the exophoria. This condition is the counterpart of that which is observed in persons with so-called relative hypermetropia, who acquire convergent strabismus when they try to correct the hypermetropia by strong accommodation. After advancement of both the internal rectus muscles the exophoria as well as the spasm of accommodation was relieved.

The patient has been under my observation for more than eleven years. Three years after the operation short attacks of spasm of accommodation recurred; they were prevented by the use of abducting prisms of 2 degrees before each eye. When the patient put the spectacles away, the spasms of accommodation recurred, combined with a convergent strabismus which gradually increased to 20 degrees. I should like to emphasize that the patient before the operation had never shown any convergent strabismus during the attacks of spasms of accommodation. Many interesting details of the observation, which I have previously published [2] must be omitted here because of lack of space. Repeated and most careful general and neurologic investigations did not reveal any pathologic condition which could be brought into causal connection with the ocular disturbance. Since the influence of psychic factors had to be considered by a psychiatrist, Professor Lange examined the patient several times. He conceded the possibility that psychic trauma participated in the origin of the anomaly but doubted the helpfulness of psychotherapeutics.

As to the ocular trouble, I was able by many examinations, made at longer or shorter intervals, to ascertain the following facts: In 1926, that is, four and one-half years after the operation, if binocular single vision was suppressed exophoria of 2 degrees for a distance of 5 meters and of 6 degrees for reading distance became manifest, the vision being normal without glasses. The range of divergence was 3 degrees and the range of convergence 10 degrees for distance. The patient for several days or weeks had binocular single vision without discomfort. At times, in particular in the evening or in a dark room when she was reading or writing, there arose crossed diplopia. She was able to overcome it, but the vision became blurred as a result of a spasm of accommodation. After an angina, the prisms of 2 degrees were no longer sufficient to produce binocular single vision, and she had to be given 6 degrees for each eye. After she had worn

2. Bielschowsky, A.: Exophorie und "Divergenzexzess," Klin. Monatsbl. f. Augenh. **92**:11, 1934.

these prisms for three and one-half years, she suffered from cystitis for several weeks. When she returned to her work, she had crossed diplopia in spite of the prism spectacles. Prisms of 9 degrees were prescribed, and in May, 1931, prisms of 12 degrees. When I saw her again in 1932 she had worn these prisms continually for near and distance vision. I found hypermetropia of 1.5 diopters and full vision. She had a range of divergence of 14 degrees for distance and 10 degrees for near vision. The range of convergence tested for distance was 10 degrees. The near point of convergence was 4 inches (10.16 cm.).

In order to find out whether the result of the bilateral advancement of the internal rectus muscle after having been perfect for ten years had finally disappeared so that the same divergent position of rest that existed before the operation had returned, I bandaged one eye for a week. When I removed the bandage in a dark room, putting a dark red glass before the one eye, the patient displayed an exophoria of only 1 degree, gradually passing over into convergence of 10 degrees. When tested at reading distance with convex glasses correcting the hypermetropia, the patient had at first convergence of 4 degrees, which gradually decreased to orthophoria and then passed over to a slight divergence, finally oscillating between 4 degrees of convergence and 4 degrees of divergence.

COMMENT

My final test proved that the abnormal range of divergence was not founded any longer on a corresponding divergent position of rest or explainable by a simple convergence insufficiency. Since the position of rest was approximately orthophoric and the near point of convergence was normal, not only the abnormal range of divergence but the relative divergence at reading distance must have been derived from an abnormal irritability of the center controlling divergence being a divergence excess of nervous origin.

As to the occasional attacks of spasm of convergence and accommodation, it must be considered that the spasms occurred only when the patient was looking at distant objects, whereas in near vision crossed diplopia always shows a lack of convergence. Before the patient was operated on a spasm of convergence had never occurred in combination with the attacks of spasm of accommodation, which is a proof that the correction of the original exophoria, which formerly had to be compensated by an increased convergence effort, still persisted. That this effort now produced a convergent squint during the occasional attacks of spasm was due to the fact that the position of rest was nearly normal.

Between exophoria and divergence excess there is a fundamental difference, exophoria being a divergent position of rest caused by definite anatomic and physiologic conditions affecting the globes and their annexes, whereas divergence excess is an anomaly of nervous origin, having nothing to do with the position of rest. Therefore, the deviation in true exophoria is nearly constant, changing only with increasing age; the deviation due to divergence excess, however, varies within

wide limits according to the momentary condition of stimulation, which depends on multitudinous and often obscure conditions. The instability of the relative position of the eyes and the alternation of divergence, convergence and parallelism of the visual lines while looking at constantly distant objects are characteristics of the spasms of divergence and convergence innervation. An analogous instability is observed in so-called double or alternating hyperphoria, which is due to intermittent excitation of the centers for positive and negative vertical divergence.

All these spasms belong to the so-called functional neuroses. A patient suffering from one of these anomalies has as a rule a neuropathic constitution, exhibiting more or less distinct symptoms of hysteria or neurasthenia. The treatment of such patients is frequently unsatisfactory unless one is able to find and remove the cause of the trouble. In this respect the convergence spasm of hysterical origin has the best prognosis because it often can be influenced by suggestive treatment. This was entirely ineffective in the reported case of divergence excess; it was just as ineffective in all the cases of vertical divergence excess that I have seen. One cannot expect satisfactory results from operative procedure in cases in which, as in the reported case of divergence excess, the anomaly is of purely nervous origin. That Duane and Dunnington obtained good results by tenotomizing the external rectus muscles proves, in my opinion, that they had to deal not with divergence excess as a true nervous anomaly but either with a divergent position of rest or with a combination of both these anomalies. In operating on a patient with orthophoria with pure divergence excess one runs the risk of producing esophoria or a postoperative insufficiency of the external rectus muscles, which causes manifest homonymous diplopia in lateral movements during the intervals between the attacks of divergence excess. In the case reported I tried prism exercises for strengthening the power of convergence. The result was unsatisfactory, as I expected, since the patient is at times capable of effecting a maximum convergence in spite of the fact that she suffers chiefly from insufficiency of convergence in near work. Because the effort that she has to make in order to produce single vision causes, as a rule, a spasm of accommodation, she resorts to the constant use of atropine and is working with convex glasses combined with prisms of 3 degrees base in.

There are some important conclusions to be drawn from these observations. First, one must distinguish a passive and an active divergence, the former being caused by an anomalous position of rest due to anatomic, mechanically acting factors and the latter by abnormal excitability or overstimulation of the center for divergence. In some cases the divergence may be caused by both factors, by the position of rest as well as by the nervous trouble. The exophoria in the reported

case was in the first period of observation either a passive or a combined passive and active divergence, whereas in the postoperative period there was a purely active divergence excess based on irritation of the nervous center.

Second, it is not admissible to infer overactivity of this center from an excessive prism divergence, since even in a case of passive exophoria kept in check by a strong fusion power an increased prism divergence is to be found, indicating that the fusion convergence relaxes in proportion to the increasing strength of abducting prisms put before the eyes. Ophthalmologists are acquainted with an analogous behavior in the gradual relaxation of the ciliary muscle when convex glasses of increasing strength are put before the eyes of a patient with hypermetropia. Only after the fusion convergence has been completely relaxed by abducting prisms will there be a slight active divergence overcoming prisms of a higher degree than necessary for compensating the exophoria. This active divergence in such cases is only a small fraction of the prism divergence measured; it amounts to about the same degree as the active divergence which is found, as a rule, in orthophoria. Therefore, to draw conclusions from the results of the prism tests is admissible only when the position of rest has already been ascertained by the other tests for heterophoria.

There are cases in which the relaxation of the fusion innervation cannot be obtained by the usual tests. For such cases Marlow and other authors recommended the prolonged occlusion test, which often gives satisfactory results but requires more time than many patients can afford. One can obtain a result as reliable as that obtained by the occlusion test in the following way: By carefully examining the duction power one determines the strongest prism which the patient is able to overcome. At the moment when diplopia appears the rotation prism is replaced by a dark red glass. A patient with exophoria will see crossed double images, the distance of which from each other corresponds initially to the angle of the strongest prism the patient had been able to overcome but very soon diminishes by a few degrees and then becomes stationary as long as the fusion tendency remains destroyed by the dark glass. This stationary deviation gives the amount of true exophoria, and the difference between this and the amount of prism divergence indicates the effect of active divergence.

DISCUSSION

DR. JOHN H. DUNNINGTON: As I see it, the author is in complete accord with Duane's ideas on the existence of a divergence center, the presence of a divergence innervation antagonistic to the convergence innervation and the innervational origin of divergence excess. The only diversity of opinion is as to the rôle that divergence excess plays in the production of divergent strabismus. It seems logical

to assume that with an innervational overactivity of divergence there must be an abnormally strong tendency for the eyes to diverge which can be overcome only by overstimulation of convergence. When through fatigue or illness the patient relaxes, the eyes diverge just as they do when fusion is interrupted. My reasoning is that with each relaxation it becomes progressively harder for the patient to maintain binocular fixation; hence in time he gives up, and concomitant divergent strabismus is the ultimate result. What part an anomalous position of rest plays I cannot say. I should greatly appreciate it if the author would go into that phase in a little more detail.

I feel confident that in spite of the innervational origin much help can be afforded these patients by receding the external rectus muscles. A considerable percentage of cases of convergence excess are of innervational origin, and they respond to recession of the internal recti. Since the case which was reported was one of a divergence excess, I cannot see why an advancement of the internal rectus was done. It seemed to me that the operation merely superimposed on divergence excess an excess of convergence. Much of the trouble might have been avoided by weakening the external rectus muscles.

DR. H. W. WOOTTON: My interest in divergence excess has been centered for the most part in cases of divergent concomitant strabismus in which the divergence is present only for distance and the near point of convergence is normal, or nearly so. In such cases the vision of both eyes is frequently normal, there is no spontaneous diplopia for distance, the refractive error is most frequently hyperopia, at least in one eye and frequently in both, and the external rotations of the eyeballs are markedly increased. In these cases of divergent strabismus (and in these only) I advocated tenotomy of one or both external rectus muscles and stated that the results had been remarkably good. My observations were published in the *Ophthalmoscope* in May 1916. I have always believed that until actual divergence commences, at which time the patient is annoyed by transient crossed diplopia for distance, a divergence excess for the most part gives no symptoms except those caused for near vision by the hyperopia which frequently accompanies it, but which I believe to have no causal relation. I have always thought that the divergence excess is caused by overaction of the divergence center, which, judging from what sometimes takes place in divergence paresis, when one external rectus muscle later becomes paretic, must be located in the region of the nucleus of the sixth nerve.

In view of the interesting and remarkable case reported this evening, which Dr. Bielschowsky had under observation for eleven years, some of my views may require modification. I have never seen a similar case, or if I have, I failed to recognize its true importance. Cases of accommodative spasm of marked degree are not rare in young adults, but I do not recollect a case associated with marked exophoria, and I did not know that exophoria could indirectly cause accommodative spasm. Does not Dr. Bielschowsky still believe that there was a strong psychic element?

DR. P. CHALMERS JAMESON: In the surgical management of divergence excess, there are certain elementary principles, namely the physical properties of advancement and recession and their potential value when applied singly under varying conditions:

1. Advancement of a muscle opposed by a strong, hypertense muscle gives a comparatively *small* degree of correction.

2. Advancement of a muscle not opposed by a markedly hypertense, hyperinnervated muscle gives a *material* correction.

3. Recession of an excessively strong muscle opposed by a sufficient or normal muscle is apt to give a *high degree* of correction.

4. Recession of an excessively strong muscle opposed by a weakly energized or muscularized muscle gives *comparatively less* correction.

5. The combined procedure, while often the best method, if performed when it is unadapted to the condition present, sometimes destabilizes the correction. In such instances, the single procedure may have been all that was necessary.

6. As a general rule, the correction obtained by producing tension or hypertension of a muscle (i. e., advancement) opposed by a muscle capable of action is much less than the correction obtained by direct relaxation of a hypertense or strong muscle.

Incidentally, in cases of divergence excess opposed by a normal muscle, a unilateral or bilateral well computed recession sometimes gives correction without accessory measures. Especially is this the case if the deviation is not of high degree.

It can be broadly stated, however, that in divergence excess the combined procedure is usually necessary, and if this is the case the question arises: On which operation, advancement or recession, shall the primary emphasis be placed? It is my plan, based on personal experience, to make the primary operation a well computed recession on the hypertense, strong muscle, bearing in mind the principle already alluded to.

The near point of convergence ascertained preoperatively is a valuable finding which points to the desirable procedure and gradation. It is worth remembering that while formerly only advancements could be computed, graded and sclerally fixed, with the recognition of the principles embodied in muscle recession and the demonstration of the safety of the scleral suture in the region of the equator, one is now able to compute, grade and sclerally fix tenotomies with as great or almost greater accuracy.

Dr. James W. White: Dr. Bielschowsky's interesting paper presents several questions for consideration, questions which will not be settled tonight but left for succeeding generations. Dr. Bielschowsky, being a scientist, is skeptical of statements until he has proved them to his satisfaction. Duane, in talking to a confrère, said "I will mistrust your findings as if they were my own." This is the true spirit of the man of research.

The position of rest has long been a problem. Le Conte stated that the normal position of rest is outward to a line bisecting the angle formed by the outer and inner walls of the orbit, or about 26 degrees. He also stated, as many have observed, that the eyes roll up when at rest and that therefore in drowsiness, sleep, intoxication and death the eyes thus roll out and up. The question that comes to my mind is whether these attitudes are the practical position of rest. Is not the practical position of rest more like a relaxed attitude taken when one is gazing into the far distance? If one considers this rather than some theoretical position, Sherrington's laws of reciprocal innervation are in force. Dr. Bielschowsky believes in a center for the control of the divergence mechanism, as do all ophthalmologists. Neurologists are not all willing to concede this point.

Dr. Bielschowsky's case, as I see it, was primarily one of divergence excess, and as a result of an illness, convergence insufficiency was added; resection of both internal rectus muscles improved the condition. When the convergence insufficiency improved with improvement in health, the operation was shown to have given an overcorrection. This seems to me to prove that Dr. Bielschowsky and Dr. Duane are trying to express the same ideas in different language.

It is unfortunate that there is not more uniformity in the testing of the muscles or, at least, that the method of making the tests is not stated. I have found a great difference in many cases, especially in those of exophoria and even of exotropia, when the tests were made by the phorometer, the Maddox rod, red glass or any of the methods depending on binocular vision as compared with the screen and parallax test. This difference has been so interesting that in many cases both types of tests are made in order to observe the difference. In some cases, however, the readings are identical. The screen test, when made slowly and with the screen held before one eye while the prisms are being changed, in my experience brings out more latent deviation than any other test except the combination of this test with the Maddox rod, as in the screen-Maddox rod test, which at times produces a still greater relaxation. In cases in which the usual tests show a possible divergence excess, the tests for distance are also made at from 150 to 200 feet (45 to 60 meters). At this distance, exotropia may be developed, while at 20 feet (6 meters) exophoria results. Marlow's occlusion test undoubtedly breaks up the fusion even more than the screen test, but aside from the inconvenience of the method, one is not sure whether a latent exophoria is fully corrected or whether, owing to so prolonged occlusion, a second factor has not been added. I have observed a few cases in which a combined convergence and insufficient divergence excess have both been increased by such occlusion.

Will Dr. Bielschowsky give his explanation of the divergence excess so frequently observed in association with vertical exophoria and exotropia? With similar findings otherwise, one patient will show convergence and another divergence. Observed cases lead one to believe that a patient will produce active convergence or divergence to rid himself of the annoyance of a vertical deviation.

Dr. Le Grand Hardy: Five years ago, in collaboration with Dr. Berens and Miss Stark, I presented at the meeting of the American Ophthalmological Society data relative to three phases of divergence excess, namely, incidence, correlation with refraction and the value of orthoptic treatment.

One hundred and two cases furnished the basis of that report, which appeared in the "Transactions of the American Ophthalmological Society" for 1929. The discussion, which I shall not read here, led to three conclusions:

1. Divergence excess occurs in the private practice of ophthalmology in from 1 to 2 per cent of all cases.

2. It is not correlated with refraction.

3. Orthoptic treatment is frequently of great value.

Since 1929 I have continued that study, and in a new series of cases the incidence of divergence excess has been between 1 and 2 per cent. In this series, hypermetropia predominates by such a small margin that the second conclusion expressed in 1929 remains unmodified. The result of orthoptic treatment in forty cases serves to substantiate the third conclusion drawn in 1929, namely, that orthoptic treatment is frequently of great value and in some cases obviates the necessity for operation and that it should be tried primarily in every case; in those cases ultimately requiring surgical intervention it should be continued as an adjuvant treatment in order to insure the best possible results.

Dr. A. Bielschowsky: The short time at my disposal makes it impossible to answer all the questions as thoroughly as I should like. As I understand it, the diversity of opinion as to the rôle that divergence excess plays in the etiology of divergent strabismus is due mainly to varying conceptions of the term "position of rest." It is not known whether there is a true orthophoria, i. e., an ideal posi-

tion of rest, in which the visual lines as well as the corresponding retinal meridians are parallel, without any fusion tendency. As a rule heterophoria occurs in the position of rest. Extensive and thorough investigations have shown that more than 80 per cent of patients began to squint after the loss of binocular vision. In about 60 per cent, the amblyopic eye deviates outward, while in a minority of cases a convergent, a vertical or a compound deviation develops. In my opinion these different kinds of deviation are due to different anatomic factors by which the position of rest in the individual cases is influenced. I have thoroughly investigated the position of rest in many hundreds of healthy people of different ages who did not complain of eye strain or other ocular disturbance. At least 80 per cent showed heterophoria; the majority, esophoria. I cannot imagine that such esophoria as well as the divergent squint developing in consequence of the loss of binocular vision is as a rule due to overstimulation of the divergence center, i. e., to an anomalous nervous excitation. The influence of nervous factors on the position of rest cannot be excluded completely, so that the rôle of the anatomic factors becomes apparent. But it is not admissible on account of this fact to deny the important rôle of anatomic factors in the evolution of divergent strabismus. Great stress must be laid on distinguishing deviations of nervous origin from those based on an anomalous position of rest due to anatomic factors, because in a case of squint of purely nervous origin, like a hysterical spasm of convergence, operation must not be performed. As soon as the spasm ceased in a case of orthophoria there would remain a latent or manifest deviation produced by the operative deviation of the normal position of rest. Therefore, I did not dare to operate on my patient in the later stage when I was convinced of the purely nervous origin of the divergence. Cases like this, in my opinion, are extremely rare. I have not seen a second case displaying the characteristics of true divergence excess, i. e., of an anomalous excitation of the divergence center. A neuropathic condition, such as was present in my patient, plays an important rôle in the various kinds of ocular spasm. The frequent occurrence of spasm of convergence is easily understood: convergence is governed by the will, whereas divergence cannot be produced voluntarily except by patients with a latent or periodically manifest divergent squint.

SWELLING OF THE NERVE HEADS WITH ARACHNOIDITIS AND UNUSUAL CHANGES IN THE VISUAL FIELDS

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I have had under observation an interesting case of swelling of the nerve heads associated with arachnoiditis and with unusual changes in the visual fields.

REPORT OF A CASE

A man, aged 41, was referred to me for refraction a little more than twenty-four months ago. At that time he gave a history of migraine which had been present for nineteen years, the attacks averaging two a month and always being accompanied by intense pain in the head, by nausea and by prostration. The pain was so severe that repeated doses of morphine were necessary. He had acquired considerable tolerance for this drug, and large doses were necessary for relief; he did not, however, use morphine except during the attacks. Corrected vision was 6/5 in the right eye and 6/15 in the left. The left eye showed an old paralysis of the inferior rectus muscle, for which he had been operated on twice. The history of it suggested traumatism sustained at birth. The amblyopia in the left eye was uncorrectable. The tension was 21 mm. of mercury (Schiötz) in each eye. The ocular motility present was typical of paralysis of the inferior rectus muscle. The fundi showed no evidences of pathologic changes save that the nerve head of the left eye was slightly pale as compared to that of the right. There were no pathologic changes in the blood vessels. Refraction showed a moderate degree of hyperopic astigmatism.

The patient returned in June 1933 stating that he was having difficulty in reading. His migraine-like headaches had become decidedly more severe and more frequent, and he was taking huge doses of morphine. He was still able to work hard all day at his office. He stated that he had been impotent for the past year. His vision was 6/6 in the right eye and 6/15 in the left with best correction. The tension was 22 mm. of mercury in the right eye and 21 mm. in the left. He needed a plus 1 sphere for near vision. There was no change in the pathologic condition of the extra-ocular muscles. The fundus of the left eye showed no apparent differences from the condition present at the time of the first examination. The right fundus, however, showed an entirely different picture. There was marked swelling of the entire temporal half of the papilla. This passed off the nerve head into the macular region. The temporal vessels were engorged and tortuous. The nasal vessels showed no change. The nasal half of the papilla was flat and had distinct margins. No hemorrhages or exudates were seen. The apex of the elevated papilla showed a central cup still present. There was a deep-seated, dark purple discoloration on the temporal side of the nerve head. The peripheral field

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Read before the College of Physicians of Philadelphia, Dec. 21, 1933.

From the Peter Ophthalmological Clinic, University of Pennsylvania, Graduate School of Medicine.

in the right eye showed a defect in the inferior nasal quadrant suggestive of a Bjerrum scotoma. In addition, there was some general contraction of the remainder of the field. The left field showed the same general contraction, with the greatest amount of loss in the nasal portion (fig. 1 *A*). There were no other signs suggesting glaucoma.

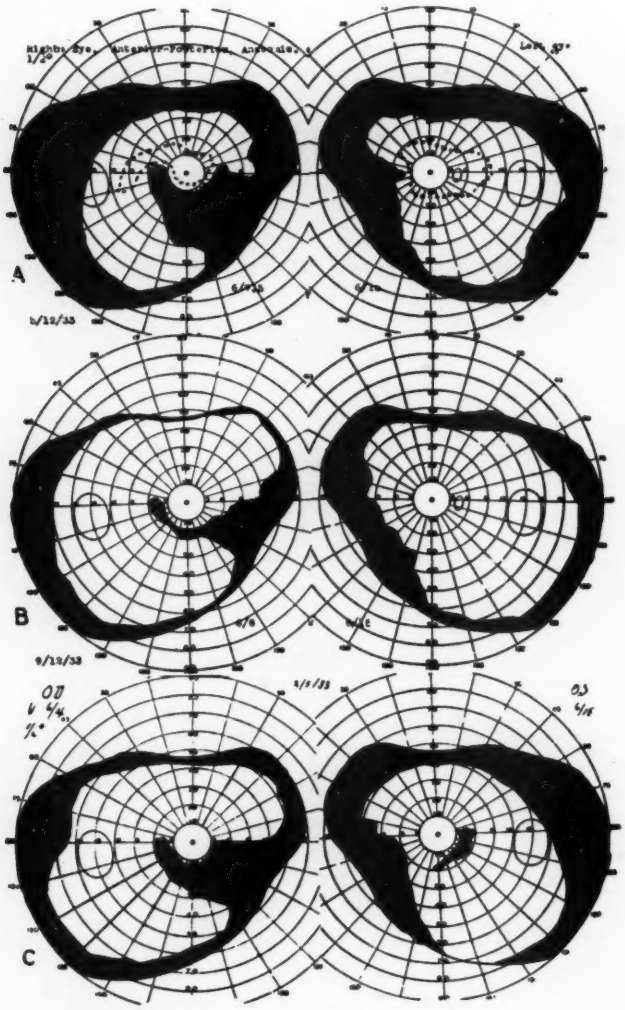

Fig. 1.—Peripheral fields before operation (*A*), immediately after operation (*B*) and seven months later (*C*).

Within two weeks there was a marked increase in the pathologic changes in the right fundus. The swelling of the nerve head continued to increase, and hemorrhages appeared about and in the papilla, limited almost wholly to the temporal half and at first rather pale. Exudates appeared about the papilla and in the macular region. The purple discloration increased so steadily in extent and in depth of color that a diagnosis of neoplasm of the optic nerve was seriously considered. No significant changes were found at physical examination, including determination of the renal function and urinalysis. Repeated Wassermann tests

gave one weakly positive reaction. The systolic blood pressure varied from 116 to 132 mm. of mercury, while the diastolic pressure remained constant at about 80 mm.

The neurologic examination revealed nothing significant except for a suggestion of weakness of the left side of the face, and even this was not certain. The spinal

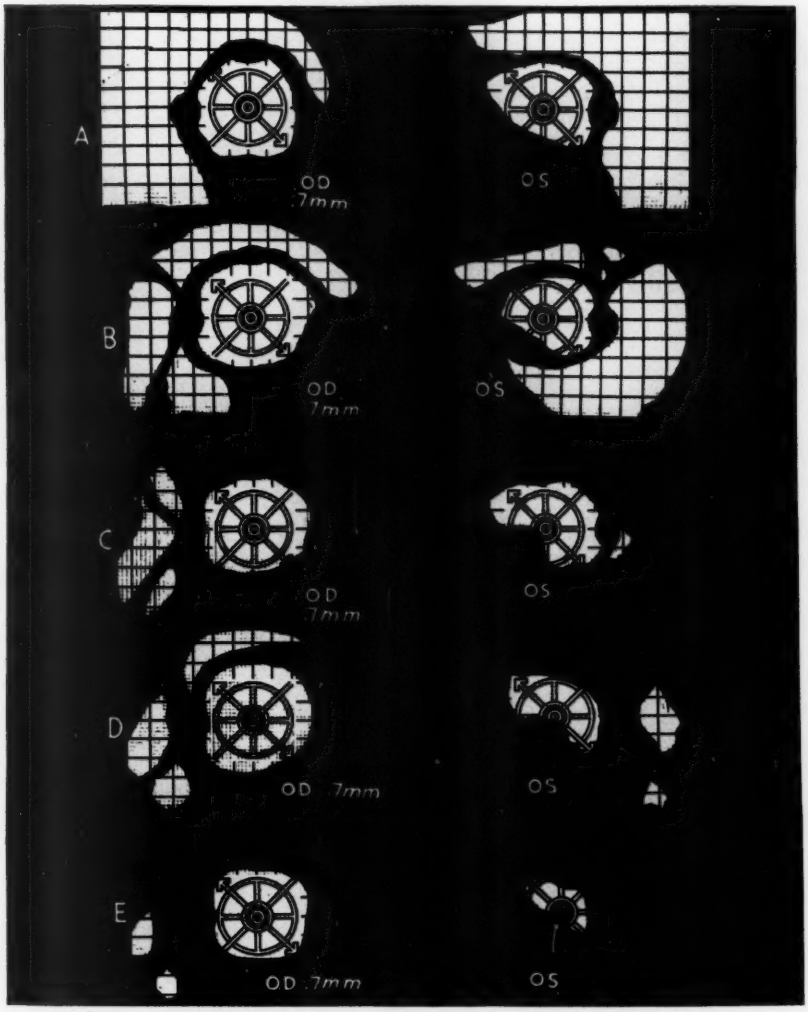

Fig. 2.—Central field studies with the Lloyd slate and with a 0.7 mm. test object.

fluid pressure was normal. The central fields with a 0.7 mm. test object showed extensive defects in the nasal portions, with enlargement of the angioscotomas. The defects represented an advance of those seen in the peripheral fields two weeks before. The angioscotomas broadened into the blind regions rather like the classic defects of glaucoma. An outstanding point of interest was the large nasal defect without visible ophthalmoscopic findings in the left eye (fig. 2*A*).

The report of the roentgen examination of the head at this time was as follows: "Both optic foramina were normal. The anterior clinoid process on the right was much more thinned and atrophied, and the anterior margins of the lesser wing of the sphenoid were a little irregular and atrophic. The tips of the posterior clinoid processes were a little more pointed than normally. There was nothing to indicate generalized intracranial pressure. With the presence of unilateral papilledema in the right eye, the peculiar cut of the field in this eye and the unusual roentgen findings, a neoplastic process involving either primarily or secondarily the optic nerve between the decussation and the emergence from the right optic foramen is to be seriously considered." The plates were taken to a second roentgenologist for interpretation, and his report, wholly independent of that of the first examiner, was almost verbatim in its similarity. Craniotomy was decided on, and a tentative diagnosis of tumor was made. Vision was 6/9 in the right eye and 6/22 in the left.

Twenty-four hours before the time set for the operation, edema appeared in the left papilla, accompanied by the same deep-seated purple discoloration found in the right eye. Here also the edema was limited wholly to the temporal portion of the papilla, but even more so to the lower inferoir temporal quadrant. Opera-

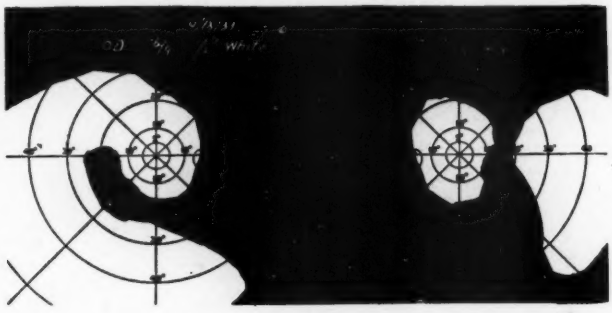

Fig. 3.—Campimetric fields immediately before operation.

tion was postponed for forty-eight hours. Hemorrhages were present after twenty-four hours, with some exudates as well. These were both limited to the papilla and to the region immediately contiguous to the inferior temporal pole of the disk. At this time the swelling of the papilla in the right fundus measured 6 diopters, and in the left fundus, about 3 diopters, in each eye the nasal half being barely elevated. The left inferior temporal vein was unusually tortuous and engorged. The campimetric fields at this time (June 18), slightly more than one month after the first observation, are shown in figure 3.

There was almost complete loss of the nasal field in each eye, with the central field still intact. It was reasonable to expect that a defect of the nerve fiber bundles complicated the angioscotoma, but there was no justification for making a differentiation of the two conditions. In view of the outcome of the case, this is significant. A vascular defect was not found in the upper field of the right eye, owing to the patient's inability to cooperate well. The right eye showed the field defect usually considered as originating from an anomalous distribution of the nerve bundles. A similar defect was present in the left eye, but to a relatively greater degree considering the duration and the extent of the pathologic process in the fundus in that eye. It showed continuation of the disproportion seen in the central fields on June 1 (fig. 1 *A*). The condition of the fundi at this time is shown in figures 4 and 5.

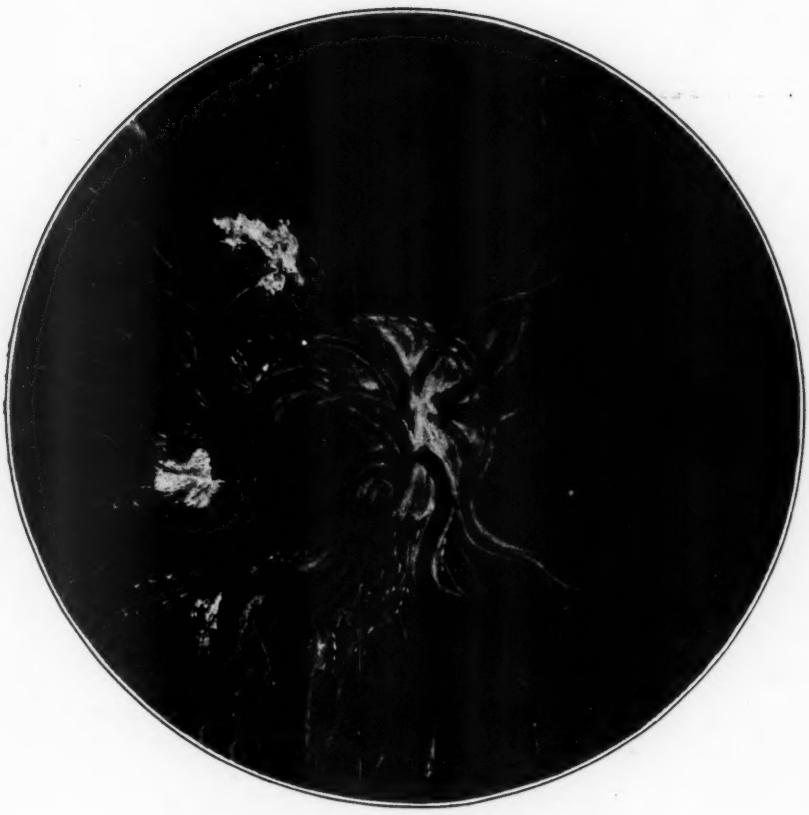

Fig. 4.—Drawing of the right fundus.

Fig. 5.—Drawing of the left fundus.

Craniotomy was done by Dr. Francis Grant and was uneventful. No neoplasm was found, but there was marked dilatation of the right anterior cerebral, the right middle cerebral and the right internal carotid artery. The vessels were adherent, with localized arachnoiditis, to the meninges, as illustrated in figure 6, and to the chiasm. The vessels on the left side also were not normal, but the pathologic changes were less marked than those on the right. The vessels bled readily when barely touched. The adhesions were released in part by dissection, the middle meningeal artery on the right was tied and the craniotomy incision was closed. The patient's recovery was a bit slow at first, but after ten days operative recovery was certain.

Eight weeks after the operation, vision was 6/4 in the right eye and 6/12 in the left. The fields at this time are shown in figure 1 *B*. There was slight but definite recession of the gross peripheral defect seen at the first examination. In

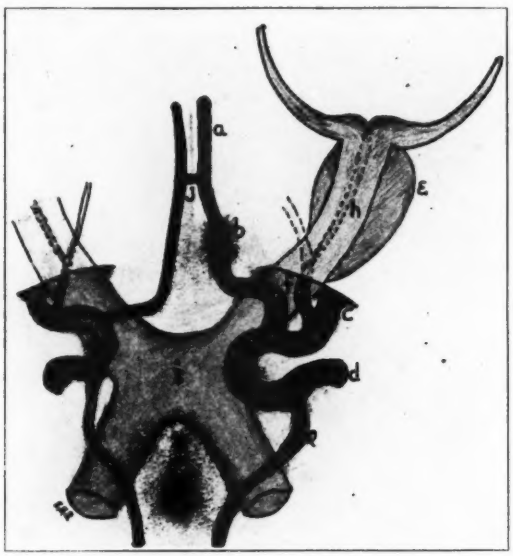

Fig. 6.—Diagrammatic drawing, to scale, of the area of pathologic change at the chiasm, as seen at operation: *a*, the anterior cerebral artery; *b*, an area of localized adherent arachnoiditis; *c*, the carotid artery; *d*, the middle cerebral artery; *e*, the posterior communicating artery; *f*, the ophthalmic artery; *g*, the dilated vaginal sheath of the optic nerve; *h*, the central artery of the retina; *i*, the optic chiasm; *j*, the anterior communicating artery; *k*, the clivus.

this interval the patient had no headaches. The fundi appeared the same as when first seen. He had taken no morphine since his immediate recovery from the operation. He was ready and anxious to return to work.

Three months after the operation he weighed 10 pounds (4.5 Kg.) more than he had ever weighed. He was back at work and felt well. His impotency had disappeared. In general, he had made a satisfactory symptomatic recovery.

Six months after the operation the patient again presented a moderate amount of bilateral papilledema, about 2 diopters in the right eye and slightly less in the left. No hemorrhages or exudates were present. The purple discoloration of the papilla was not seen. The edema involved the entire papilla equally. The vision

was unchanged. The peripheral fields were as charted in figure 1 *C*. The central fields, taken with a 0.7 mm. test object, are shown in figure *2 B*. They again revealed marked general contraction and a nasal loss similar to that shown in the earlier chart but with more extensive defects. In this chart the angioscotomas appear most typical. It seems justifiable to consider that the greater portion of the gross field loss was due to impairment of the nerve fiber bundles. The patient had no general symptoms except for rather obstinate constipation. At this time the light sense threshold was less than one half of the normal.

Two weeks later the papilledema had disappeared, as had also the constipation, perhaps as a result of a diet outlined to correct it. Four weeks later papilledema reappeared and again was more marked in the right eye. Three weeks later the nerve heads were once more fairly normal in appearance. The central fields taken at this time are depicted in figure *2 C*. Continued impairment was evident.

The central fields have remained fairly stationary for some time. Figure *2 D* shows the appearance four weeks after the last determination referred to. The left field remained as a bare central island, and that on the right was a bit better preserved. At the time that these fields were taken, a second determination was made with pressure applied over the carotid artery, first to one side and then on the other. In each instance the pressure was held for two minutes, direct pressure being applied through a roll of gauze bandage held so firmly that the pulsations of the carotid artery were transmitted distinctly to the roll. There is no doubt that some blood was flowing past this interference, but for the short time of the test it could have been but little, for in each instance the patient was definitely light-headed and had a brief period of dizziness. This was done to determine, if possible, what damage would follow to the field should ligation of the carotid artery be carried out later. These central fields are shown in figure *2 E*.

This question could not be answered from the findings since collateral circulation, which it was hoped would develop, could nullify the transient manifestations shown. Certainly, however, there was a brief period of retinal anemia with venous stasis and with brief minor retinal edema. Evans [1] and Turner,[2] separately, have discussed the effect of edema in sufficient detail that further mention herein would be superfluous. There is little doubt that if the pressure on the vessel had been continued the entire field on the left soon would have been wiped out, certainly the remaining portion of the central island, and the same process seemed to be occurring in the right eye, though not as rapidly. It is not unusual that this case, which illustrates so well both angioscotomas of pathologic size and defects in the nerve fiber bundles should thus corroborate some of the work which has already been done on this subject. I regret that I cannot go into greater detail relative to this interesting phase.

Coincident with the field changes illustrated in figure *2 D*, the light sense threshold became raised to less than one fourth of the normal in the right eye and to one sixth of the normal in the left, but it has remained at that level up to the time of this report. A slight but significant discrepancy is present, however. The field in the right eye has widened a bit, while that in the left eye has remained about the same, but the light sense threshold has shown further impairment in each eye.

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1. Evans, J. N.: The Scotometry of Retina Edema, Am. J. Ophth. **16**:9 (May) 1933.

2. Turner, A. H.: The Circulatory Minute Volumes of Healthy Young Women in Reclining, Sitting and Standing Positions, Am. J. Physiol. **80**:60 (May) 1927.

The probable outcome of this case is problematical. Fractional ligation of the internal carotid arteries may be necessary sooner or later. The aneurysm-like deformity of the vessels may continue to increase and death occur by rupture. A satisfactory prognosis seems impossible.

If material should become available later histopathologic study would be valuable. Recently Saphir [3] presented a histopathologic study of the optic nerves and chiasms of six patients with a similar condition and concluded "that visual disturbances are not likely to follow from the subsequent pressure exerted upon the nerve, for the microscope shows only slight changes in the neural tissue." While my patient shows no true sclerotic changes (as yet), he certainly shows and has shown definite effects from damage to the optic nerves and to the chiasms. Therefore, I feel free to differ from Saphir in his conclusion. A sufficient number of analogous situations also furnish adequate reasons for my attitude. Further, the literature has already presented cases of visual defects resulting from direct pressure on blood vessels, and it must not be overlooked that the immediate cause of arteriosclerotic atrophy of the optic nerves seen with the ophthalmoscope is not always clearcut. Abelsdorff [4] stated that this has been definitely proved. The differences in opinion are interesting.

It is possible in considering this case to draw some rather interesting inferences and to discuss some probabilities which may be of value. The cessation of the attacks of migraine may be due to one of two different factors. The first, the decompression, could be only temporary; its mechanism is unknown, and its value would continue only until the vessels had become reattached to the surface of the chiasm in a new position. There they would once more start damage through their exaggerated, and hence pathologic, pulsations. The second, and more probable, factor is the ligation of the middle meningeal artery.

There is little doubt that the dark purple discoloration which appeared in each papilla was due to a slow but steady extravasation of blood along the vaginal sheath of each nerve and between its fibers until it reached the sclera. The hemorrhage probably came from the ophthalmic artery and certainly accounted for some of the less distinct retinal hemorrhages extending through the vaginal sheath, for all of them did not come from the central retinal vessels. Those from the central retinal vein as the result of pressure and of stasis (?) appeared darker, had distinctly outlined limits or margins and were not on the papilla itself. The hemorrhages appeared earlier than did the exudates.

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3. Saphir, O.: Changes in the Optic Nerves Resulting from Pressure of Arteriosclerotic Internal Carotid Arteries, Am. J. Ophth. **16**:2 (Feb.) 1933.

4. Abelsdorff: Atrophy of the Optic Nerve Through Arteriosclerotic Closure of the Arteria Centralis, Ztschr. f. Augenh. **52**:273, 1923.

The perimetric findings are of outstanding interest. The changes in the fields are evidently due to impairment of the nerve fiber bundles in the optic nerve anterior to and at the chiasm and to disturbances of the vascular supply of the optic nerve pathway and of the retina. They simulate the fields of glaucoma rather closely, perhaps as a result of the same causes as found in glaucoma, i. e., the damage to the individual nerve bundles and circulatory disturbances, which occur especially in glaucoma simplex. In a case of glaucoma the fibers are damaged before they leave the eyeball, while in this case the fibers were almost undoubtedly affected after they left the eyeball and as they lay in the optic nerve and in the chiasm.

Such field changes are not new, though they have not previously been reported in this type of case. Bjerrum [5] stated, in discussing the scotoma which bears his name:

> I have seen visual fields similar to those of glaucoma described in other diseases, though without the ordinary pressure atrophy of the optic nerve. In neuroretinitis hemorrhagica one can find visual fields that are typically glaucomatous, and also in some cases of partial embolism of the arteria centralis retinae. It is not surprising that a disease which localizes itself more or less in the edges of the papilla should present fields with a similar topographic localization and resemble those which I (Bjerrum) have described. A disease which is present at the edge of the papilla or in the walls of its excavation should present similar fields, regardless of whether it is glaucoma or some other pathologic condition, such as retinitis hemorrhagica which is attacking the optic nerve.

Salzer,[6] in discussing glaucoma as a disturbance of circulation, spoke of these and of like defects coming from the blind spots and later changing into large nasal defects as not being necessarily characteristic of glaucoma. He found them without glaucomatous manifestations in diseases of the nasal accessory cavities, in choked disk, in optic neuritis, in papillary and retinal hemorrhages, in myopia, in choroiditis and especially in simple arteriosclerosis. Seidel, according to Chirkouski,[7] showed in a discussion at the Heidelberg conference in 1924 that the narrower defects which seem to stretch from the place of departure at the blind spot are not characteristic of glaucoma. On the basis of his investigations, Samoilov came to the conclusion that the

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5. Bjerrum: Scandinav. Ophth. Magazine **5**:132, 1892.

6. Salzer: Glaukomale Kreislaufstörung, München. med. Wchnschr. **75**:768 (May 4) 1928.

7. (*a*) Chirkouski: Concerning the Significance of Campimetry for Investigations of the Function of the Eye in Its Physiological and Pathological States, Russk. arch. oftal., 1929, p. 354. (*b*) Dymschitz, L. A.: Campimetry and Its Importance for the Investigation of the Functions of the Eye in Its Physiological and Pathological Conditions, ibid., 1929, p. 354.

ribbon-like changes are the result of lymphatic disturbances in the perivascular spaces of the vessels of the retina. Chirkouski stated:

> This point of view (Samoilov's) is of undoubted interest with regard to the question of the pathogenesis of Bjerrum's scotoma. In general, it should be observed that the significance of vascular defects (angioscotomas) originating in the blind spot has not been up to the present time sufficiently appreciated. And yet they are of great significance and generally related to the "Nervenfasern-defekte."

This circumstance was pointed out by Hess in his criticism of dot perimetry. He stated "that it is quite probable that in some cases the defects of vascular origin are really typical Bjerrum scotomata."

When one looks at the charts as shown and knows the pathologic changes found in this case, it is likely that these two factors, the angioscotomas and the nerve bundle defects, are wholly independent. It is likely, as well, that the former do not change into the latter; the normal radial position of the angioscotomas corresponds to a sufficient extent with the whorl of the retinal fibers to simulate but not to cause defects in the nerve bundles. It is permissible to suspect that they may influence the development of nerve bundle defects because of simple mechanical causes similar in part to those which lie at the base of the development of such defects, but this has not been proved. One or both may be present in the same case at different times or at the same time. Evans [1] stated:

> Much more evidence derived from well controlled studies must be accumulated. We know certain things quite definitely about fiber bundle defects in general. For instance in lesions of the optic pathway in its retrobulbar course, sector defects are classical, and as a general rule have their apices pointing toward the macula. On the other hand, defects known to arise from retarded fluid flow, both blood and lymph, are characterized by more irregular defects, which however have a tendency to form sectors with the apex of the defect pointing toward the blind spot. This of course omits central scotomata from consideration, but probably does not except the so-called concentric contractions which very often, upon careful analysis with minute objects, will be found to be a blending of obtuse angle sectors with bases running together and apices toward the enlarged blind spot. The defects known to arise from vascular disturbances represent a wide variety of pathological entities which include such as arterio-sclerosis, nasal accessory sinusitis, intracranial pressure, etc.

This has been mentioned earlier in this paper. Evans further stated:

> Since there is no doubt that there are both fiber bundle damage and vascular disturbances in every case (glaucoma), at some time in its course we must accept the idea that the defects met with in the disease may have their origin—some by one mechanism, some by another. Though many have conceded that the vascular disturbances precede even the pressure rise, it behooves us to give careful attention to the recent elaborate study of Salzmann, who, by building up microscopic pro-

jections, has reconstructed the progress of the venous occlusion in the vessels as they pass through the nerve head.

The size of the normal as well as of the pathologic angioscotoma in terms of perimetric degrees does not correspond necessarily to the ophthalmoscopically visible size of the corresponding vessel or vessels. Other factors must enter into the extent of this formation. The angioscotoma may be microscopic, biochemical, physical but macroscopically invisible or of some other type. Nerve fiber defects do not demand a direct increase of intra-ocular pressure for their formation; they developed in my case from indirect extra-ocular pressure applied to the nerve trunk itself. Certainly the arachnoiditis, the unusual hemorrhages, the swelling of the nerve heads and the absorption of bone prove the presence of local damage to the chiasms and the nerve trunks.

The various studies of the central fields illustrated rather well a point frequently overlooked in regard to the physiology of the retina. It is an error to consider the retina as a fixed, standardized, sensitive photographic plate always ready for the reception of incident rays and for subsequent cerebral interpretation of the stimuli aroused by these rays. One must consider it instead a sensitive membrane but a labile one, wherein there is a constant "flow like that of the to and fro movement of the tide" (Evans), dynamic and not static, ever changing and influenced tremendously by a fluidity of the biochemical structure, which must be present but which is subject to as many different physical factors as is any fluid in any portion of the body. Vision is by no means a pure function of the rods and cones in their fixed mosaic-like fabric of the retina; other factors less well known have an equal importance.

CONCLUSIONS

Any given case of binasal hemianopia should be carefully investigated before it can be considered a genuine case of this rare defect. Fields no more typical of pure damage to the nerve fiber bundles than those shown in this case have been reported as representing binasal hemianopia. The case reported presents unusual fields, which under conditions of greatest damage simulate binasal hemianopia, and may progress to such a degree of binasal field loss that later in the course true differentiation will be impossible. Figure 7 shows two pairs of fields from Lutz'[8] analysis of binasal hemianopia. The upper fields are from one of thirteen cases reported in his review which I feel are probably similar to mine. In all of these thirteen cases the patients had generalized and localized (intracranial) vascular disease. The lower fields rep-

8. Lutz, A.: Über binasale Hemianopsie, Arch. f. Ophth. **119**:423, 1928; Ueber einige weitere Fälle von Binasaler Hemianopsie, Arch. f. Ophth. **125**:103, 1930.

resent true binasal hemianopia. Although in the case presented here the greatest loss lies in the nasal halves of the field, the case is no more one of true binasal hemianopia than is a similar one wherein retinitis pigmentosa was considered the underlying cause.

Angioscotomas and defects in the nerve bundles are very likely independent, certainly early in any given case. It appears that visual field changes attributable to pathologic variations of the angioscotomas are later modified by associated nerve bundle defects, and also that this can arise in cases of intracranial vascular disease with arachnoiditis through modification of the circulatory or vascular supply of the brain and the retina.

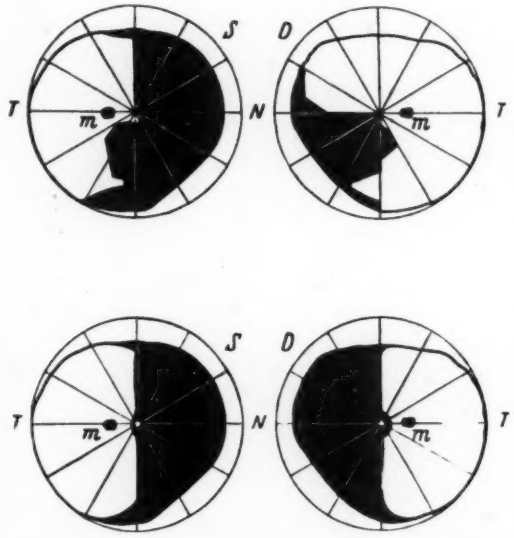

Fig. 7.—Nasal hemianopia as described by Lutz. The upper fields are from a case of postneuritic optic nerve atrophy with sclerosis of the retinal vessels. Vision was 6/7 in the right eye and 6/10 in the left. There was normal color vision. The lower fields are from a case of bilateral papilledema with intracranial neoplasm and show absolute central scotomas. Vision was 1/200 in the right eye and 1/100 in the left.

If papilledema should, as I have stated in another communication, be unaccompanied by a definite rise in the light sense threshold, this patient did not have true papilledema. His fundi, on the other hand, did not present papillitis. The central fields also illustrate the marked relationship which exists between perimetric findings, as illustrated, and the light sense threshold, or simply the light sense from the detailed portions of the retina. In fact, Evans, in discussing Marlow's paper on the diagnosis of glaucoma simplex, stated: "It was quite apparent that properly conducted examinations of the visual fields with reduced

illumination and with relatively minute objects may be interpreted in terms of a light sense test, the diagnostic value of which is proven by the importance we have come to assign to visual field studies." I feel that examination of the central fields, with minute test objects, tells what will be found later when using an arc perimeter under the usual standard conditions and methods of peripheral field investigations.

The Seidel sign and the Bjerrum scotoma, so often considered as the onset of nerve bundle defects, may be in many instances angioscotomas of pathologic size, as they seem to appear in my case. Further, they are not, of necessity, diagnostic of glaucoma. Considering the field defects found in this case with known vascular pathologic changes and comparing them with the defects found so frequently in glaucoma, one is, of necessity, impressed by the great rôle that the retinal circulation (both blood and lymph) must play in the symptomatology of glaucoma.

OPHTHALMOMYIASIS SUBRETINALIS

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In May 1932 Dr. van Waveren enucleated the left eye of a peasant boy from Koegras (the Netherlands). Particulars concerning the case follow.

REPORT OF A CASE

History.—A boy, aged 3 years, was the youngest of five children. There was no history of ocular disease in the father, the mother, the other children or any of the relatives. Nothing peculiar about the patient's eye was noticed until Christmas of 1931, when the parents were struck by the child's "dull expression." He had not had an accident or any infectious disease and did not complain of pain in the eye.

On May 17, 1932, the right eye was normal; even after maximal dilation of the pupil and careful examination, no abnormalities were observed in the fundus.

The position of the left eye was normal; the lids, conjunctiva and cornea were normal; there were no keratic precipitates or circumcorneal congestion. The anterior chamber was somewhat shallow. The aqueous humor was clear. The iris was moderately wide. The pupil was moderately wide, oblique, oval and undilatable by atropine. There were no nodules. The temporal half of the iris bulged out and showed marked atrophy of the pigment layer. The position of the other half, which likewise showed local depigmentation, was normal. The entire iris had a greenish hue in comparison with that of the normal eye. In the bulging part of the iris there were a few dilated vessels. Ash-gray, nonpigmented membranes surrounded the pupillary margin. The crystalline lens was perfectly clear, as well as the vitreous body just behind the lens. The fundus reflected a yellowish-green light. A tumor could be observed, three fourths of which protruded into the vitreous body. The intra-ocular tension was slightly raised. A diagnosis of pseudoglioma or glioma of the retina was made. On May 23 the eyeball was removed.

Microscopic Examination.—The bulb was sent to our laboratory for microscopic examination by my associate and me. After the usual fixation and hardening it was cut in horizontal sections 0.02 mm. thick in pyroxylin. We began by examining a section containing the optic nerve; the pupil was not yet visible. We observed episcleral infiltration, localized especially around the ciliary vessels. The anterior as well as the posterior segment showed considerable changes (fig. 1).

Only slight changes were observed in the cornea. The epithelial layer was normal; the membranes of Bowman and Descemet showed no particular changes. The substantia propria was perfect in structure, although it contained vessels. A remarkable observation was the abundance of eosinophilic cells in the periphery of the cornea; these cells could also be seen around the vessels. The anterior chamber was reduced to a small space, bounded by the normal cornea in front and

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From the Department of Ophthalmology of the University of Amsterdam (Director, Prof. Dr. W. P. C. Zeeman).

by the anterior layer of the iris behind. It was filled with coagulated material containing chiefly lymphocytes as well as a great number of eosinophilic leukocytes.

The iris, i. e., the iris stroma, was for the greater part situated, beginning at its insertion, against the back of the cornea. Near the center it bent back to the anterior surface of the normal lens, simulating an "iris bombé." This condition, however, was not present, as a part of the pigment layer adhered to the capsule

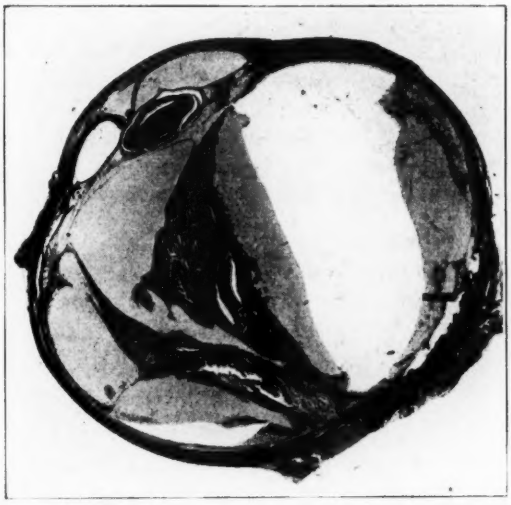

Fig. 1.—Section containing the optic nerve, showing changes in the anterior as well as in the posterior segment.

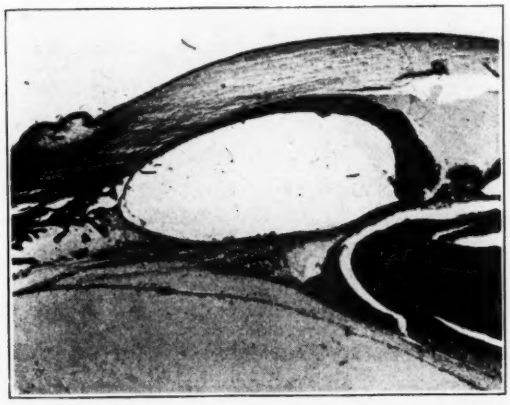

Fig. 2.—Section showing a cyst of the pigment layer of the iris.

of the lens, thus forming a so-called total posterior synechia. The pigment layer therefore was split (fig. 2). The iris was greatly changed; it showed a marked increase of vessels and locally circumscribed infiltrations of lymphocytes and plasma cells, as well as a great many eosinophils. The ciliary body was comparatively flat, suggesting myopia, and was infiltrated. On the interior unpigmented epithelial layer of the thin ciliary processes, "sprouts," such as are observed in cases of chronic iridocyclitis, could be seen.

This case furnished a perfect example of a cyclitic membrane as described by Straub, with obliterated vessels and Treacher Collins' "glands of the ciliary body." The posterior chamber, which was considerably reduced in connection with the total synechia, was filled with various blood cells and bounded by the cyclitic membrane. This membrane continued along the posterior surface of the lens in a newly formed tissue composed partly of compact fibrous tissue and partly of material of looser consistency, with hollows containing erythrocytes. Considerable changes were observed in the posterior part of the eye. The retina was entirely detached from the layer underneath, extending like a funnel from the disk toward the lens. A formation of fibrous tissue could be observed in its folds. There was distinct perivasculitis retinae accompanied by infiltration. The subretinal space was partly filled with coagulated fluid in which there were many loose cells and conglomerates of cells. The choroid was highly hyperemic and showed considerable dilatation of vessels with excessive diffuse infiltration, which assumed a more focal character here and there.

COMMENT

From these facts one may draw the conclusion that there was an extensive inflammation in the whole bulb, accompanied by exudation of cells, which were chiefly eosinophilic leukocytes. Fuchs,[1] as well as Brückner,[2] has pointed out that eosinophilia in the eye is rare. It is well known that eosinophilia is especially observed in conjunctivitis in the form of spring catarrh; it is also observed with hay fever, sporotrichosis and Pascheff's acute necrotic conjunctivitis. Weinberg[3] succeeded in causing eosinophilia by instilling ascaris extract into the conjunctival sac; it has also been observed in cases of intra-ocular cysticercus.

In the present case the patient suffered from none of the diseases mentioned. It naturally occurred to us that the inflammation must be due to an unusual cause, and our surmise proved to be correct. When examining the other sections, we were struck by a peculiar structure in the subretinal space, which was observed in fifty-nine sections (fig. 3). This consisted of a thin, homogeneous outer layer, which appeared to have been much damaged by cutting, surrounded by a quantity of exudate cells, among which there were a great many eosinophils and erythrocytes. Elements were seen in this outer layer which were without doubt not of human origin.

Dr. de Meyere, professor of entomology at the University of Amsterdam, who examined the microscopic preparations, expressed the opinion that the structure in question was a segment of a Diptera larva. In the center of the obliquely cut larva there was a hollow lined with bulb-shaped, vesicular, chromaffin cells, which was doubtless the

1. Fuchs, E.: Ueber Eosinophilie im Auge, Arch. f. Ophth. **95**:162, 1918.

2. Brückner, A.: Cytologische Studien am menschlichen Auge, Arch. f. Ophth. **100**:179, 1919.

3. Weinberg, M., and Séguin, P.: Recherches biologiques sur l'éosinophilie, Ann. Inst. Pasteur **28**:470, 1914.

intestinal tract of the larva. Broad-based, chitinous hooks were distinctly visible on the surrounding cuticula (fig. 4 *A*). A number of striped muscle fibers were also observed, easily distinguishable from peculiar dendritic formations, which must be considered as the tracheal tubes. In some places there were chains of cells; these were ganglion cells (fig. 4 *B*).

This case was, therefore, one of ophthalmomyiasis interna. Until a short time ago the occurrence of parasitic larvae in the human eye was considered to be a great rarity and was apparently almost exclusively observed in the tropics. During the last ten to fifteen years, however, some cases have been recorded in the ophthalmologic literature in Europe, principally in Russia and the Balkan states, as well as a few in England, Germany and France. A case in the Netherlands was reported by Zeeman [4] in 1926, and Zeeman has shown considerable

Fig. 3.—Section showing the subretinal larva. In the center the intestinal tract may be seen.

interest in the study of the second case, that reported here. Behr [5] was the first, in 1920, to attempt to define ophthalmomyiasis as a separate syndrome. He distinguished between ophthalmomyiasis externa, in which fly larvae are found in the conjunctival sac, and ophthalmomyiasis interna, in which the parasites are found in the eye. Ophthalmomyiasis externa has often been observed and described. In 1931, Kreiker [6] described a case in a man aged 20. An insect flew against his left eye. A few minutes later the eye began to smart, and in his endeavor to locate a foreign body the patient himself discovered small moving worms on the conjunctiva. Within ten minutes he was in a clinic, where yellowish-white maggots of 1.5 mm. in length were seen in the eye, which fled from the light into the fornix. Syringing and eversion of the lids were of no avail, as the larvae attached themselves

4. Zeeman, W. P. C.: Nederl. tijdschr. v. geneesk. **70**:1192, 1926.
5. Behr, C.: Klin. Monatsbl. f. Augenh. **64**:161, 1920.
6. Kreiker, A.: Klin. Monatsbl. f. Augenh. **87**:262, 1931.

firmly with their hooks. It was only after liquid petrolatum had been instilled into the conjunctival sac that the air passages and the trachea became choked up and the larvae were suffocated. More than thirty larvae were removed in this way. The next day the patient had entirely recovered. Ticho,[7] of Jerusalem, described six cases of ophthalmomyiasis externa which he had observed. All the descriptions have one symptom in common, namely, that shortly after an insect flew against the eye, acute conjunctivitis developed, often attended

Fig. 4.—*A*, section of the larva showing the chitinous hooks on the cuticula. *B*, section showing muscle fibers (*m*), ganglion cells (*g.c.*), the trachea (*tr.*) and the intestinal tract (*i.t.*).

with the formation of follicles, and it was always possible to remove from the conjunctival sac the often numerous larvae, either alive or killed, without causing permanent damage to the eye.

That ophthalmomyiasis externa may lead to ophthalmomyiasis interna is proved by Maggiore's [8] statements. A peculiar white spot the size of a pinhead was observed in the cornea of a boy of 16. At

7. Ticho, A.: Brit. J. Ophth. **4**:177, 1923.

8. Maggiore, L.: Larva di dittero nella camera anteriore: II. Contributo all' ophthalmomyiasis, Ann. di ottal. e clin. ocul. **6**:67, 1922.

first it was impossible to make a diagnosis. The spot developed inward, with increasing symptoms of inflammation, and in a few weeks a white object was observed in the anterior chamber, which proved to be a larva. In 1909, a case was reported by Thomas and Parsons,[9] in which the maggot of a blow fly or a sarcophagous insect lay across the anterior chamber on the surface of the iris. It had given rise to intense iridocyclitis, with detachment of the retina, and the eye had to be enucleated.

A larva has also been found sometimes in the posterior part of the eye. Von Hess,[10] in 1913, was the first to find a fly larva behind the

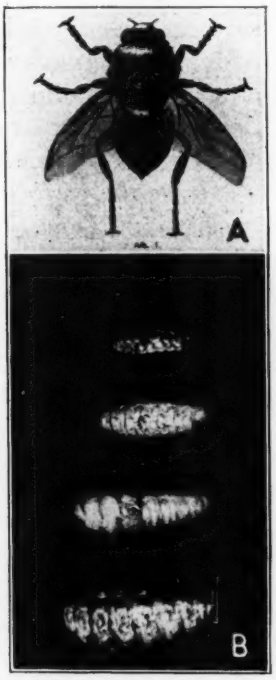

Fig. 5.—*A*, Hypoderma bovis. *B*, larvae of Hypoderma (actual size).

retina. Cases in which a subretinal fly larva was observed, examples therefore of ophthalmomyiasis interna subretinalis, were afterward published by Schmidt zu Wellenburg [11] in 1917, by Behr [5] in 1920, by Zeeman [4] in 1926, and by Archangelsky and Braunstein [12] in 1931.

9. Thomas, F. G., and Parsons, J. H.: Tr. Ophth. Soc. U. Kingdom **29**:14, 1909.

10. von Hess: Arch. f. Augenh. **74**:227, 1913.

11. Schmidt zu Wellenburg, H.: Centralbl. f. prakt. Augenh. **41**:1, 1917.

12. Archangelsky, W. N., and Braunstein, N. E.: Zur pathologischen Anatomie der Ophthalmyiasis interna, Klin. Monatsbl. f. Augenh. **87**:340 (Sept.) 1931; Russk. oftal. j., 1931, vol. 14.

A perusal of the different descriptions of ophthalmomyiasis interna reveals a marked concurrence of symptoms, making it almost possible to construct a typical syndrome of this disease. In all the aforementioned cases of ophthalmomyiasis interna in which the larvae were examined by entomologists, they were supposed to be Diptera larvae of the Oestridae family, probably larvae of Hypoderma bovis, or the hornet (fig. 5). Dr. de Meyere came to the same conclusion in this case. Dr. Baudet, curator of the Institute for Parasitic and Infectious Diseases in Utrecht, sent me a number of H. bovis larvae at different stages of development, which I examined microscopically. The hornet flies during the summer months and lays its eggs on hot days on the limbs of cattle. The larvae coming out of these eggs, which are a few millimeters long, pierce the animal's skin and after a long journey through the body finally arrive after six months under the skin of the back, having grown to a length of several centimeters. Young larvae are sometimes found even in the walls of the esophagus of cattle as well as in the spinal cord. The full grown larvae leave the worm lumps and pupate on the ground, developing into insects within from twenty to thirty days.

Of course the question arises as to how the larva penetrates into the eye. In reviewing the different cases of ophthalmomyiasis interna, one finds that all the patients were under the age of 10 years, which is probably not a coincidence. Maggiore's 16 year old patient should be regarded as an exception. In hardly any of the cases mentioned was an insect sting observed near the eye, and the parents of our patient maintained on being carefully questioned that they had not noticed a pimple or swelling of the face. It seems improbable that the children transferred the larva to the eye in one way or another by means of their hands, as according to published reports these creatures pierce the skin very rapidly. Brauer, for instance, said that when he placed an Oestrus larva on his arm it vanished under the skin before he had time to take up his magnifying glass. The most probable explanation is that the ova are deposited near the eye and the larvae rapidly penetrate into the tissue, generally without being noticed; they make their way to the eyeball by piercing the outer sclera. The circumstance that ophthalmomyiasis is observed only in young children is accounted for by the fact that the sclera of an adult is difficult to pierce, having become harder and thicker with age, while a child's soft, thin sclera offers little resistance. Parsons has expressed a similar opinion. To my regret it was impossible in the case reported here to determine the *porte d'entrée* with any degree of certainty. Zeeman [4] was able to reconstruct the probable route of the larva in his case by study of the sections. It appeared to have penetrated to the eyeball under the upper lid, through the conjunctiva and along a ciliary vessel. Behr [5] was also able to indicate, clinically as well as anatomically, the diascleral course followed by

the larva in the case described by him. Archangelsky and Braunstein [12] arrived at the same conclusion.

Highly improbable is the opinion held by the first investigator, von Hess,[10] that the larva came into the eye through the circulation of the blood. The H. bovis larva is practically the exclusive cause of ophthalmomyiasis interna. The other larvae found in the conjunctival sac, for instance that of O. ovis, follow another course of development and lack the faculty of being able to pierce comparatively hard tissues.

As a rather characteristic symptom of ophthalmomyiasis interna should be mentioned the intermittent course of the disease, which consists of a severe follicular conjunctivitis, ciliary injection, opacity of the cornea, iritis, photophobia and lacrimation. If the larva has penetrated deeper, an apparent conjunctivitis phlyctenularis may appear, attended with scleritis and followed by intra-ocular symptoms, such as hemorrhage into the vitreous, cyclitis, choroiditis and dialysis retinae, often giving the impression of a pseudotumor.

Our little patient had every chance of coming into contact with hornets; the hygienic conditions in his home were poor. There were cattle in the vicinity, and in addition, according to eyewitnesses, the place swarmed with flies. The initial symptoms remained unnoticed probably because in such surroundings little attention is paid to minor complaints.

ADDITIONAL REFERENCES

Nanes: Ann. d'ocul. **159**:130, 1922.
Villard, H.: Ann. d'ocul. **159**:670, 1922.
Wright, R. E.: Am. J. Ophth. **10**:411, 1927.
Linke, O.: Graefes Arch. **126**:644, 1931.

ACQUIRED CYSTS OF THE SCLERA

M. N. BEIGELMAN, M.D.
LOS ANGELES

Epithelial cysts of the eye, particularly the intra-ocular ones, have been the subject of numerous and extensive studies. The fundamental principle involved in the formation of these cysts, the proliferation of surface epithelium within the deeper lying tissues, has been established early and is applicable to any of the ocular structures involved. However, depending on the part of the eye invaded by epithelium, the clinical aspects of the resulting cystic formation vary to a considerable extent. Cysts arising in the anterior chamber, in the iris, in the cornea or in the sclera differ materially in appearance, course and significance for the affected eyeball.

In the sclera epithelial cysts are observed comparatively seldom. Since 1853 twenty cases of this type of cyst have been reported by seventeen authors, three of whom—MacKenzie,[1] Lauber [2] and Mattos [3] —described two cases each. In the last few years I have had the opportunity of studying three instances of this uncommon disease; in two the wall of the cyst was examined histologically, and in one the cyst was removed and sectioned with the eyeball in toto.

REPORT OF CASES

CASE 1.—Mrs. T. Z., aged 28, came to my office in July, 1929, complaining of pain in the left orbit and of inability to wear an artificial eye in it. She stated that she had a penetrating injury to the left eye about fifteen years before. Following this, the globe decreased in size, and eventually she was able to wear a prosthesis. In the last few years the patient noticed a slowly growing "tumor" on the shrunken globe.

On examination the left eyeball was found in a state of phthisis. The cornea was small, flattened and entirely opaque. An old retracted scar could be seen at the upper part of the limbus. There was, directly above it, a large bulging cystic formation, with the conjunctiva freely movable on its surface. The wall of the cyst was grayish white, thin and translucent; on palpation one gained the impression of an easily fluctuating mass (fig. 1). Enucleation was advised.

Owing to the size of the cyst, a canthotomy was performed preceding the enucleation. Aside from this the dissecting, the severing of the muscles and the sectioning of the optic nerve presented little difficulty.

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1. MacKenzie, W.: Diseases of the Eye, London, 1854; quoted by Lauber.[2]

2. Lauber, H.: Ueber Skleralcysten, Arch. f. Ophth. **58**:220, 1904.

3. Mattos, W. B.: Traumatic Cysts of the Sclera, Rev. ophth. São Paulo **1**:107, 1931.

In the specimen thus obtained two parts could be readily discerned (fig. 2): The lower, about one fourth or one fifth of the whole, was solid and represented the shrunken eyeball; attached to it above was a large cyst with a thin wall which gradually, almost imperceptibly, blended with the sclera of the phthisic globe.

When the specimen was cut in two and examined with a loupe, it became obvious that the large cystic formation was situated entirely within the sclera. No communication leading from this cyst into the eyeball could be detected. The

Fig. 1 (case 1).—Large scleral cyst.

Fig. 2 (case 1).—Enucleated eyeball. The smaller lower part represents the shrunken globe; the larger upper part, the scleral cyst

globe itself was shrunken and deformed, with a piece of bone embedded in its wall. An unusual feature of this phthisic eyeball was the presence of a centrally located cavity which resembled the scleral cyst in the appearance of its lining.

After the usual fixation, dehydration and embedding the specimen was examined in serial sections.

The topographic relations, as found in the microscopic sections, were rather complicated. A schematic drawing (fig. 3) shows them in a simplified manner. The lining of the scleral cyst consisted of epithelial cells, greatly flattened and

arranged in from two to four layers. In some places the epithelium appeared to be markedly thickened, up to six and even eight layers. Through an examination of the adjoining sections it was established that this "thickening" was merely the result of an oblique sectioning. The connective tissue of the cystic

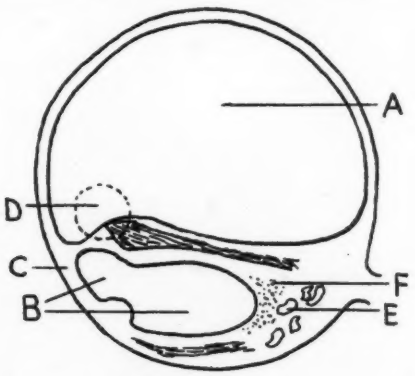

Fig. 3 (case 1).—Schematic drawing of the specimen in figure 2. *A*, intrascleral cyst; *B*, intra-ocular cyst; *C*, cornea; *D*, wedgelike incarceration of ciliary body (the encircled area is shown in figure 4); *E*, ossification of the choroid; *F*, probable remains of the retina.

Fig. 4 (case 1).—Wedgelike incarceration of the ciliary body between the scleral pillars. This was probably the starting point in the formation of the scleral cyst.

wall differed little from ordinary scleral structure; it was thinner and poorer in nuclei. Inside of the cyst occasional epithelial cells in various stages of degeneration and some red corpuscles were found.

In the phthisic eyeball the corneosclera was represented by a band of dense connective tissue. This fibrous coat appeared to be markedly thickened, not

only because of a "relaxation" taking place in any shrunken globe, but because it was reenforced by newly formed organized material of inflammatory origin. There was no difference between the structure of the cornea and that of the sclera. Only the surface epithelium on one side of the globe and the entrance of the optic nerve on the other permitted the localization of the anterior corneal and posterior scleral portions of the fibrous coat. Corresponding to the corneoscleral junction a wedgelike incarceration of the ciliary body was seen (fig. 4). This was the only part of the uveal tract that was fairly well preserved and

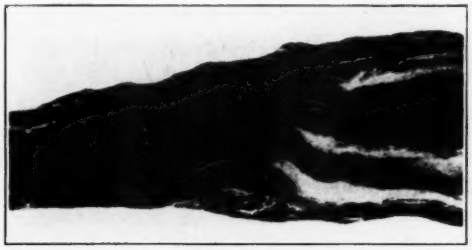

Fig. 5 (case 2).—Wall of a scleral cyst

Fig. 6 (case 3).—Wall of a scleral cyst.

still recognizable in outline. The remainder of the uvea could be identified only through the presence of pigment. In a number of sections a bony plate was found closely connected with a deformed choroid. Some large collections of poorly stained nuclei, situated well within the globe, possibly indicated the folded and distorted remains of the retina. The intra-ocular cyst mentioned in the description of the macroscopic specimen could be seen in the form of a large cavity lined by one or two layers of flattened epithelial cells and occupying the place of both the anterior chamber and the vitreous.

Case 2.—Mr. J. R., aged 52, was examined in the outpatient department of the Los Angeles County General Hospital on April 6, 1932. The patient had a small (2 by 2 mm.) cystic formation attached to the sclera of the right eye, with the conjunctiva freely movable in this area. The cyst was located at the outer part of the limbus, almost exactly in the horizontal meridian. The patient

was not aware of the cyst and could give no information as to its origin. He had had repeated minor injuries and inflammatory conditions of the eye in childhood.

On April 13, 1932, an attempt was made to excise the cyst after exposing it through a conjunctival incision. Only a portion of the wall could be removed, and this was subjected to microscopic examination. The specimen was found to consist of scleral lamellae lined by one to three layers of epithelial cells (fig. 5).

I saw this patient several months after the operation. At the place of the cyst there was a marked indentation in the sclera.

CASE 3.—Mr. W. D., aged 79, was seen in the outpatient department of the Los Angeles County General Hospital on July 7, 1930. He had senile cataracts in both eyes and a small cyst of about 2 mm. at the limbus of the left eye. This was considered a cystic "pinguecula." In removing the "pinguecula" on July 27, 1932, it was noticed that the cystic formation was "fixed to the underlying tissue." However, owing to its small size it was possible to excise the cyst in full. When examined under the microscope the specimen was found to be a multilocular cyst with from two to six layers of flattened epithelium on its inner surface (fig. 6). The wall of the cyst was formed by bundles of connective tissue fibers of varying thickness and closely resembled the scleral structure.

COMMENT

Among the twenty-three cases of scleral cysts reported to date a definite history of injury was given in seventeen cases. It is interesting that in this group five cysts developed following operations on the globe (iridectomy, extraction of a cataract or evisceration). Of the remaining six cases two, in spite of a history to the contrary, presented on histologic examination direct evidence of trauma: Lawrence [4] found in the posterior wall of the cyst an opening leading into the eyeball, while Rubert [5] detected particles of pine needles in the affected area. Besides, de Vincentiis [6] mentioned a burn of the eyeball preceding the formation of the cyst, and Rados [7] gave the history of an ulceration. It is obvious that the great majority of acquired scleral cysts should be considered as traumatic, particularly if the scope of trauma is widened to include any impairment of the integumentary structures.

The surgical cases in which the patients remained under continuous observation following operative procedures on the eyeball provide definite information regarding the period of time necessary for the develop-

4. Lawrence, W.: A Treatise on Diseases of the Eye, London, 1853; quoted by Odinzov: Vestnik oftal. **27**:996, 1910.

5. Rubert, J.: Zur Kenntnis der intrascleralen Zysten, Klin. Monatsbl. f. Augenh. **48**:145, 1910.

6. de Vincentiis: Convention of the Italian Ophthalmologic Society, Turin, 1887, Zentralbl. f. prakt. Augenh. **11**:340, 1887.

7. Rados, A.: Ueber spontane Iriscysten und traumatische Scleralcysten, Arch. f. Ophth. **99**:152, 1919.

ment of a scleral cyst. Gruening,[8] Pravosud [9] and Filatov [10] stated that they noted the first signs of cystic formation in the sclera several months after the operation. From the history of the cases with injury one gets the impression of a considerably longer space of time intervening between the original injury and the onset of scleral changes. This is due to the fact that in the absence of subjective symptoms the earlier stages in the development of a scleral cyst escape the patient's attention. One usually becomes aware of the (scleral) deformity only when it reaches the stage of being conspicuous or troublesome.

Once the cyst comes into existence its further progress varies from case to case. In a few instances the scleral cyst remained almost stationary, not exceeding 2 or 3 mm. in diameter. More often it grew slowly, attaining within a few years the size of a bean or walnut, and in exceptional cases the cyst became eventually larger than the eyeball itself.

During the process of continuous distention the anterior wall of the cyst is likely to become thinned, thus changing the consistency of the new formation from some degree of solidity to a soft, almost fluctuating, type. At the same time the translucence of the scleral cyst may become evident on simple inspection, while in the earlier stages it calls for the use of a powerful and well focused illumination (slit lamp). Sometimes the posterior wall of the cyst also yields to increased pressure, and as a result of the uveal pigment shining through the thinned scleral structures a dark tint is added to the appearance of the cyst (Eisenstein's [11] case).

Owing their start to trauma, the scleral cysts are always situated in the anterior part of the eyeball, mostly at the limbus. On several occasions, having penetrated into the cornea proper, they have been described as corneoscleral cysts. More often, however, the limbus forms a natural boundary for the growth of a scleral cyst, and the latter is likely to encircle a part of the cornea, assuming the shape of a kidney or a bean.

It follows from this description that scleral cysts are sufficiently characteristic in their appearance to be recognized without difficulty in spite of their rarity. A review of the literature reveals that on one occasion this condition was confused with a subconjunctival cyst. The

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8. Gruening, E.: A Case of ‚Sclero-Corneal Cyst, Tr. Am. Ophth. Soc. **9**: 339, 1900.

9. Pravosud, R. G.: Demonstration of a Patient with a Cystoid Scar, Vestnik oftal. **24**:260, 1907.

10. Filatov, V. P.: A Case of Cysts of the Sclera and of the Iris, Russk. oftal. j. **2**:122, 1923.

11. Eisenstein, J.: Ein Beitrag zur Kasuistik der Scleralcysten, Ztschr. f. Augenh. **19**:92, 1907.

differentiation is based on the relation of the conjunctiva to the underlying cyst: In the case of scleral cysts the conjunctiva is freely movable above them, while in subconjunctival or episcleral cysts it constitutes the anterior wall of the new formation. In another instance a scleral cyst was first diagnosed as staphyloma. The history of an injury, the consistency, the translucence and the absence of a characteristic bluish color usually permit the ruling out of a scleral ectasia.

If the problem of clinical diagnosis in the case of scleral cysts is far from being involved or intricate, that of pathogenesis presents many uncertainties and deserves a more detailed consideration.

Of primary importance in this respect are the histologic observations. Unfortunately, in spite of the fact that material for pathologic study was available in practically every instance of scleral cysts, six of the reported cases do not contain any reference to the microscopic changes. In seven cases in which surgical intervention was limited to a removal of the anterior wall of the cyst examination of the tissue was only of diagnostic significance, having revealed bundles of scleral lamellae with an epithelial lining. Attempts were made in some of these reports to establish whether the epithelium was of conjunctival or of corneal origin. While under normal circumstances such differentiation can be easily made, the epithelium proliferating within the sclera is likely to modify its appearance under the influence of new nutritional and mechanical factors. Because of this, in many instances, the origin of the cyst epithelium, the only point of pathogenetic interest in this type of material, could not be definitely determined.

A much more valuable group consists of cases in which the whole eyeball was examined histologically, particularly when serial sectioning was resorted to. This method permitted a complete topographic study of the specimen with a view to establishing the exact relation of the scleral cyst to other parts of the eyeball.

The importance of these reports justifies reviewing them at the present time, more so because several of them escaped the attention of previous contributors. Only the cases of Michel,[12] Früchte and Schürenberg[13] and Ishreyt[14] have been repeatedly referred to and will therefore be briefly mentioned here. Rados'[7] interesting case reported by him first in 1919 has been thoroughly overlooked in all subsequent articles dealing with scleral cysts until it was recently described again

12. Michel: Ueber seltene Befunde in verletzten Augen, Ztschr. f. Augenh. **6**:1, 1901.

13. Früchte, W., and Schürenberg: Ueber Korneo-Skleralzysten, Klin. Monatsbl. f. Augenh. **44**:404, 1906.

14. Ishreyt, G.: Ein Fall von intrasclerale Zyste, Klin. Monatsbl. f. Augenh. **45**:59, 1907.

by him [15] in the ARCHIVES. No credit was ever given to Gruening,[8] although he was the first one to present, in 1901, a complete study of a scleral cyst removed with the eyeball. De Lapersonne's [16] case, published the year preceding the World War, has been entirely ignored. Finally the two cases of Russian authors (Odinzov [17] and Potechina [18]) remain unknown outside of the country where they were originally reported.

At the thirty-sixth annual meeting of the American Ophthalmological Society Gruening [8] presented "A Case of Sclero-Corneal Cyst," in which the microscopic study was made by G. S. Dixon. The patient underwent an iridectomy at the age of 12. Shortly afterward the eye became much changed by the appearance of a "tumor," and two years later a cystlike growth encircling three fourths of the periphery of the cornea was found. The eye was then enucleated and sectioned. The cyst consisted of two parts; the length of the larger sac was 19.5 mm.; that of the smaller one, 13.5 mm. Stained sections showed that this formation extended from the sclera into the cornea. The walls of the cysts were found lined irregularly by a thin layer of endothelium. On careful inspection two small canals were revealed, passing from the posterior chamber into the larger cyst. The uveal tract was markedly atrophic; the retina was detached.

In Michel's [12] case several intercommunicating cystic spaces were found in the corneosclera. They were lined with one layer of epithelium, and remains of Descemet's membrane and of uveal tissue could be seen in the septums between them.

Lauber [2] described a multilocular cyst in the sclera with epithelium of conjunctival origin. There was an opening in the posterior wall of the cyst, forming a communication between the latter and the interior of the eyeball.

Früchte and Schürenberg [13] reported two separate cysts of the sclera in an injured eye. They were lined by stratified epithelium. The posterior wall of each cyst was entirely intact.

In Ishreyt's [14] case the scleral cyst, situated at the limbus, was small and contained watery fluid and a number of cells.

Odinzov's [17] patient was operated on for cataract at the age of 12. On examination eight years afterward a bean-shaped cystic formation

15. Rados, A.: Traumatic Epithelial Cysts Within the Eye, Arch. Ophth. **9**: 392 (March) 1933.

16. de Lapersonne, F.: Kystes de la sclérotique par inclusion epithéliale traumatique, Centralbl. f. prakt. Augenh. **37**:265, 1913.

17. Odinzov, V. P.: The Implantation of Epithelium and the Formation of Intra-Ocular and Scleral Epithelial Cysts, Vestnik oftal. **27**:996, 1910.

18. Potechina, E. A.: A Case of Scleral Cyst after Evisceration of the Eyeball, Russk. oftal. j. **9**:485, 1929.

1 by 2 cm. was found in the sclera concentric with the external half of the cornea and starting from the operative scar. The eye was painful; the intra-ocular tension was increased, and the vision was reduced to perception of hand movements at 0.5 meter. The enucleated eyeball was sectioned serially. No communication could be detected between any of the five cavities which formed the scleral cyst and the interior of the eyeball. The walls of the cyst were lined by epithelium of the conjunctival type. Several small cystoid areas with the same kind of epithelium were found in the anterior chamber.

De Lapersonne's [16] case was that of a young woman who had had an injury to the eye at 3 years of age. Some years afterward a cystic formation appeared at the limbus of that eye. When the patient was 19 the cyst occupied the entire lower nasal quadrant of the eyeball and consisted of five lobes of unequal size. On pathologic examination a cyst measuring 5 by 9 mm. was found in the anterior part of the sclera, encroaching somewhat on the cornea. A small amount of blood was present in the cavity. In studying the full series of sections, de Lapersonne came to a level at which the continuity of the scleral fibers was interrupted by an invagination of uveal tissue. Inside of the globe small cystic diverticula were found lined by epithelium of the same type as that found in the scleral cyst.

Rados [7] had the opportunity of examining a shrunken eyeball which contained an enormous scleral cyst. The inner surface of this cyst was covered by two or three layers of epithelium. It was not possible to determine whether these epithelial cells had originated from the cornea or from the conjunctiva. Rados described a small communicating hole between the inner hollow space of the cyst and the interior cavity of the eye. In addition an epithelial cyst was found in the anterior chamber of the phthisic eye. The lining of the latter cyst retained all the properties of the limbus epithelium. It is of particular interest that Rados was able to follow up on his sections the gradual penetration of the surface epithelium through the corneosclera into the anterior chamber.

A unique case, important from the pathogenetic standpoint, was reported by Potechina.[18] Four years after an evisceration of the eyeball which was performed on account of absolute glaucoma, the patient returned with a cystoid formation attached to the sclera of the eviscerated eye. The stump was enucleated. On examination of the specimen a large cyst (10 by 10 mm.) was found in the scleral wall of the shrunken mass. The cyst was multilocular; it consisted of several intercommunicating cavities. Stratified epithelium of the conjunctival type could be seen on its inner surface.

These pathologic data in conjunction with some of the established clinical facts form a basis for the discussion of the possible pathogenesis

of scleral cysts. Since the cysts are defined as cavities lined by epithelium and filled by liquid substance, the problem can be naturally divided into two questions: (1) the origin of the epithelial cells and (2) the source of the cystic fluid.

As was brought out before, practically all acquired cysts of the sclera are the result of trauma. The healing of any wound in the corneosclera involves an active and, to a certain extent, excessive proliferation of the surface epithelium. Under ordinary circumstances this overgrowth of epithelium, the "epithelial cone," is eventually taken care of by a reactive inflammation around it; the superfluous epithelial cells degenerate, break up and disappear entirely. The situation changes when a primary union of the scleral wound becomes impossible because of some mechanical interference. The obstacle may appear in the wound posteriorly in the form of prolapsed uveal tissue, an incarcerated lens capsule, and so forth; the anterior lips of the scleral wound may be kept apart through the inversion of a conjunctival flap or through the penetration of a foreign body. This enables the surface epithelium to penetrate quite deeply and to line the traumatic slit in the sclera. When the scleral wound is finally closed, a small epithelial cyst is already in existence within the sclera. A bridge of epithelial cells connecting the surface with the cyst could undoubtedly be found if cases of this kind were investigated pathologically in earlier stages. Many years after the injury, when the specimen becomes available for microscopic study, this connecting link of epithelial cells is no more in evidence, having disappeared in the process of cicatrization. One will recognize in this line of reasoning Stölting's [19] classic conception of intra-ocular epithelial cysts, applied to conditions existing in injuries of the sclera.

Another possible manner of formation of scleral cysts is the implantation of surface epithelium in accordance with the theory developed for intra-ocular cysts by Rothmund [20] and Buhl. Through an impact of the foreign body some epithelial cells may become dissociated from the adjoining ones and be transferred into deeper parts of the sclera. Then, instead of a direct spreading of the surface epithelium into the wound, an independent and self-sustaining proliferation of displaced epithelial cells occurs. Most investigators agree (Szekely [21]) that the Buhl-Rothmund theory has only a limited application in regard to the so-called "pearl cysts" or epidermoid cysts in the anterior chamber.

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19. Stölting: Die Entstehung seröser-Iriscysten, Arch. f. Ophth. **31**:99, 1885.

20. Rothmund: Zur Pathogenese der Iriscysten, Klin. Monatsbl. f. Augenh. **9**:397, 1871.

21. Szekely, J.: Ein Beitrag zur Genese der Epithelauskleidung des Augeninnern (Kammercysten), Arch. f. Ophth. **130**:52, 1933.

However, in the only experimental work done on scleral cysts, Niwa [22] was able to produce them by placing particles of conjunctiva or cornea in scleral pockets. He failed to accomplish similar results with foreign bodies such as gum or wood. When using particules of bulbar conjunctiva, Niwa found goblet cells in the wall of the resulting scleral cyst. The success obtained in 38.46 per cent of all the animals operated on cannot be ignored when estimating the rôle of direct epithelial implantation in the origin of scleral cysts. Neither should it be overlooked that, at least in reference to the conjunctiva, a direct carrying in of the epithelium during an injury and the resulting formation of an epithelial cyst have been proved conclusively by Mayou.[23] In his case only one week elapsed between the injury and the pathologic examination. This length of time was insufficient to permit a disappearance of epithelial strands connecting the cyst with the conjunctival surface if these were present at all. Nevertheless on serial examination "no process of epithelium could be found in any of the sections connecting the surface epithelium with that of the cyst." Mayou felt justified in assuming that his was an implantation cyst.

Greater difficulties arise when one seeks to determine the source of fluid which constantly accumulates in the original scleral slit and distends it into a cavity of appreciable size. In few instances was there a communication between the cyst and the interior of the eyeball. In the absence of direct channels for the influx of intra-ocular fluid, a transudation from the uveal tract into the scleral cyst may have taken place, provided some uveal tissue was incarcerated in the scleral wall. This theory proposed by Michel [12] is not applicable to the many cases in which the cyst was situated entirely within the sclera and was walled off from the uvea by solid connective tissue. Most interesting in this respect is the case reported by Potechina [18] in which a large scleral cyst developed following evisceration of the eyeball, in other words, while there was neither a uveal tract present nor any intra-ocular fluid available. In these instances at least, the sources of the fluid must be considered in the immediate locality of the cyst, in the sclera proper. Rubert was of the opinion that a circulatory derangement in the traumatized part of the sclera may lead to an accumulation of lymph which then participates in the formation of the cyst. It must also be mentioned that in the process of extensive epithelial proliferation the central cells may degenerate, turn into mucoid material and later become liquefied in

22. Niwa, I.: Experimentelle Studien über die traumatische Cyste der Sclera und des Ciliarkörpers, Acta soc. ophth. jap. **34**:80, 1930; quoted in Zentralbl. f. Ophth. **25**:249, 1931.

23. Mayou, M. S.: On Implantation Dermoids of the Conjunctiva, Roy. Lond. Ophth. Hosp. Rep. **16**:318, 1906.

analogy with the successive changes described by Oatman [24] for "epithelial cystomas of the conjunctiva." Finally the capacity of epithelial cells to carry on their original functions when transferred into a different tissue should be recalled. It is possible that the goblet cells of the bulbar conjunctiva continue their secreting activities within the newly formed scleral cavity and provide it with some of the liquid contents. An important point which may throw light on the source of the cystic contents is their chemical nature. This should be considered by future investigators, as in the past the matter of chemical analysis in vivo has been thoroughly overlooked. As an example, one may take the work done by Usher [25] in a case of congenital cyst of the limbus. In that case a qualitative and quantitative study established that the components of the cystic fluid were essentially those of blood lymph.

The anatomic and physical peculiarities of the sclera have a direct bearing on certain phases in the general problem of the pathogenesis of scleral cysts. It has been emphasized by Rados that because of its limited blood supply scleral tissue is a rather inadequate medium for epithelial proliferation, particularly when compared with the strongly vascularized iris. This, no doubt, accounts for the relative infrequency of scleral cysts. Another point of interest is the effect of structural changes in the sclera due to age on the formation of cysts. According to Krekeler [26] the juvenile sclera is rich in cells, which are larger and less flattened than in later years; no deposits of calcium between the scleral fibers are found at this age. These anatomic facts indicate that the juvenile sclera is likely to offer less resistance than the firmer and denser fibrous coat of older patients. It should not be surprising, therefore, that scleral cysts are mainly a disease of the young. In a few cases the cysts developed late in life, and in these instances there was either a severe and extensive trauma (exenteration of the eyeball in Potechina's case) or an extremely small cystic formation (cases 2 and 3).

Owing to a varying degree of resistance in different parts of the corneosclera, the distention of a growing cyst appears to follow some definite tendencies. The spreading takes place anteriorly until it reaches the limbus, which under ordinary conditions constitutes a barrier for further cleavage. Only when the injury occurs at the corneoscleral junction is there a possibility of the cyst's extending into both the sclera and cornea. Ishreyt's assertion that the course of scleral fibers changes at the attachment of the recti muscles in the sense of greater compact-

24. Oatman, E. L.: Epithelial Cystoma of the Conjunctiva (Dermo-Epithelioma of Parinaud), Arch. Ophth. **33**:113, 1904.

25. Usher, C. H.: Epithelial Cyst of the Sclera, Brit. J. Ophth. **10**:177, 1926.

26. Krekeler, F.: Die Struktur der Sclera in den verschiedenen Lebensaltern, Arch. f. Augenh. **93**:144, 1923.

ness and resistance to splitting carries with it the weight of his competence in problems of scleral structure. However, both in the case of Rados and in case 1 of this report this boundary had been overstepped by a rapidly growing cystic formation.

The management of scleral cysts does not present any serious difficulty. The choice of a therapeutic method depends on the condition of the globe in which the cyst has formed. If as a result of the injury the eye becomes totally and permanently blind, it is is best to exenterate or to enucleate the eyeball. The latter procedure can, as a rule, be carried out in the usual manner. A canthotomy preceding the enucleation, for the purpose of better exposure, is advisable only in the presence of an exceedingly large cyst. When the visual functions are preserved, intervention must be limited, of course, to the cyst alone. Complete excision of the cyst is not possible for technical reasons: an attempt would involve a difficult and unsafe scleral dissection. Neither is it necessary. The mere removal of the anterior wall of the cyst has proved in the past to be an entirely adequate measure.

TUMOR OF THE ORBIT IN A CASE OF OSTEOCHONDROFIBROSARCOMATOSIS

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Primary sarcomas occur with great frequency among tumors of the orbit. Birch-Hirschfeld [1] collected the reports of 1,566 cases of orbital tumors, 710 of which were sarcomas. By far the greater number of these were the round cell types and fibrosarcomas. Instances of chondrosarcoma,[2] myxochondrosarcoma [3] and myosarcoma [4] are reported, but they are rare. According to Peters,[5] osteosarcomas occur much more often than tumors containing cartilage or muscle. Sarcomas of metastatic orgin are rare. Those in the reported cases were found to have their primary source, respectively, in the mediastinum,[6] the retroperitoneal tissuse,[7] the lung,[8] the intestine,[9] and the femur.[10] The last mentioned case is the only instance in which the primary tumor affected osseous tissue. Rollet and Colrat [11] described several instances of metastases in the orbit from sarcomas of the suprarenal gland, in one of which the tumor was found to invade the frontal sinus. In the case of Meiggs and de Schweinitz [6] in which autopsy was obtained, the metastatic growths were found to affect the contents of the orbit as well as

From the Laboratories and Ophthalmological Division of Mount Sinai Hospital.

1. Birch-Hirschfeld, A., in Graefe-Saemisch: Handbuch der gesamten Augenheilkunde, Leipzig, Wilhelm Englemann, 1920, vol. 9, pt. 2, p. 712.

2. Pokrowsky: Ztschr. f. Augenh. **32**:401, 1914.

3. Van Duyse: Klin. Monatsbl. f. Augenh. **51**:264, 1913.

4. Weeks, J. E.: Report of Two Cases of Metastatic Carcinoma of the Choroid and One Case of Myxosarcoma of the Orbit, Tr. Am. Ophth. Soc. **14**: 326, 1915.

5. Peters, A.: Orbita, in Henke, F., and Lubarsch, O.: Handbuch der speziellen pathologischen Anatomie und Histologie, Berlin, Julius Springer, 1931, vol. 11, pt. 2, p. 463.

6. Meiggs and de Schweinitz: Am. J. M. Sc. **108**:193, 1894.

7. Bietti, A.: Metastatisches endotheliales Sarkom im Musc. rectus inferior, Klin. Monatsbl. f. Augenh. **54**:462, 1915.

8. Buendia: Rev. méd. German, iber. Am. **3**:869, 1926.

9. Samuels, B.: Some Notes on Orbital Tumors, Arch. Ophth. **7**:868 (June) 1932.

10. Bride, T. M.: Tr. Ophth. Soc. U. Kingdom **49**:462, 1929.

11. Rollet and Colrat: Arch. d'opht. **41**:583, 1924.

the muscle cone. However, in no case have metastatic sarcomatous deposits been demonstrated in the bones of the orbit.

In the case reported here, marked proptosis of the left eye was caused by a mass arising from the great wing of the sphenoid and compressing the contents of the posterior portion of the orbit. The tumor was a rapidly growing fibrosarcoma containing, in addition, immature cartilage and bone. However, it was but one of numerous malignant growths arising from various parts of the skeleton and finally resulting in death.

REPORT OF A CASE

History.—R. D., a man, aged 27, was apparently in good health until five weeks prior to admission to the hospital. His illness began with pain in the dorsal region of the spine. Two weeks later, although the pain subsided somewhat, numbness developed in the toes of the right foot. A few days later the numbness extended to the left thigh, so as to involve the entire left lower extremity. With the development of the numbness, there appeared progressive weakness of both legs, so that a week before admission to the hospital the patient was no longer able to move them. Throughout this time he complained of stiffness of the neck.

Physical Examination.—There was a tender mass on the left fourth rib, with tenderness on pressure over the fourth dorsal vertebra; the sphincter ani muscle was relaxed; there were paresis of both upper extremities, more marked on the left side, flaccid paralysis of both lower extremities and various other neurologic signs indicating a compressive lesion of the spinal cord in the upper dorsal region.

Lumbar puncture revealed bloody cerebrospinal fluid, but there was no apparent block in the flow of the fluid. A second lumbar puncture done a week later revealed complete block. Twelve days after admission, an exploratory laminectomy and decompression from the seventh cervical to the third dorsal vertebra were performed. No tumor was seen, but the cord was found to be swollen. The immediate postoperative course was uneventful. On the eighth postoperative day, twenty days after admission, it was noticed that the tumor nodule in the left fourth rib became larger and, in addition, a tumor nodule was discovered over the left fifth costochondral junction. There was a swelling of the right zygoma. New tumor nodules appeared over other ribs. Despite the fact that no tumor was found at operation, it was the consensus that the lesion of the spinal cord was due to compression by a tumor. A biopsy specimen from the nodule over the left fourth rib revealed fibrosarcoma. Repeated search for Bence-Jones protein in the urine gave negative results. Secondary anemia developed. The hemoglobin was 72 per cent and there were 3,510,000 red blood cells. The patient received roentgen therapy (four doses) over the area of the tumors. The general course, however, was rapidly and progressively downward, and three months after the onset of illness the patient died.

Examination of the eyes showed the following: On admission the pupils were dilated, but reacted to light and in accommodation. The fundi were normal. Three weeks later the left eye became prominent and lateral, and upward gaze was limited in this eye. There was paralysis of the left external rectus muscle. The exophthalmometer reading on the right eye was 20.5 mm. and on the left eye, 27 mm. During the further course of the illness the exophthalmos progressed practically to proptosis. No masses could be palpated within the margins of the orbit.

Autopsy.—When the brain was removed from the skull, a hard, grayish, nodular mass was seen to occupy the roof of the left orbit. This mass arose from the great wing of the sphenoid and projected somewhat farther back than the normal posterior edge of the wing. In the left orbit was a tumor mass arising from the roof of the apex corresponding to the mass in the interior of the skull. No other masses were found in the orbit.

The entire orbital portion of the left optic nerve was obtained and fixed in formaldehyde. The posterior portion of the nerve was distinctly flattened owing to compression by the orbital growth. Its anterior third appeared normal. In addition, the posterior segment of the globe was removed and fixed in Bouin's solution.[12] Paraffin sections were prepared; staining was done with hematoxylin and eosin, and with Weigert's and Kulschitzky's stains for medullary nerve sheaths.

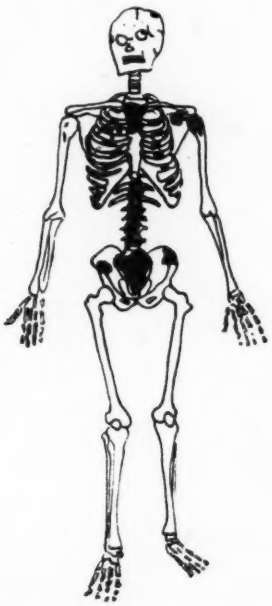

Fig. 1.—Diagram indicating areas (in black) in which tumor masses were demonstrated at autopsy.

Diagnosis.—The diagnosis was osteochondrofibrosarcomatosis of all the vertebrae and of the ribs, skull, sternum, pelvic bones, left humerus and scapula. The masses for the most part were firm, white, flat tumor nodules. In the sternum they were mulberry-shaped and were harder and bony in consistency. The masses on the ribs were often as large as a peach, while along the anterior and lateral surfaces of the vertebral column were numerous plum-sized, irregular, fungating masses, attached to the vertebral bodies and infiltrating the paravertebral muscles. The sacrum was entirely ensheathed by a firm, white tumor mass forming a box-like enclosure at the lower end of the spinal column. In both iliac fossae were large irregular tumors arising from the iliac bones. Section of the sacrum showed

12. This solution is made up as follows: solution of formaldehyde, U. S. P., 500 cc.; distilled water, 1,500 cc.; glacial acetic acid, 100 cc., and trinitrophenol to the point of saturation.

irregular thickening and destruction of the cortex with replacement by the tumor tissue. In addition, there were: metastases to the lungs, right auricle and perirectal tissue; an isolated nodule in the mesentery of the descending colon; compression of the spinal cord by a tumor of the vertebrae with myelitis; ascites; hydrothorax, and splenomegaly.

Fig. 2.—Drawing of the anterior portion of the interior of the base of the skull. *T* indicates the tumor arising from the left great wing of the sphenoid; *TS*, a mass arising from the body of the sphenoid on the right side.

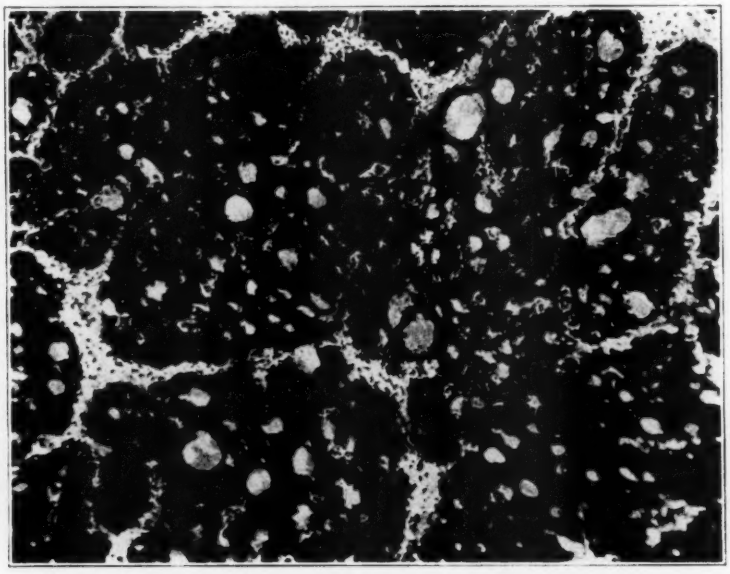

Fig. 3.—Section of the posterior part of the intra-orbital division of the left optic nerve showing cystic areas of degeneration. Kulschitzky's stain.

Microscopic Structure.—Sections taken from the paravertebral, thoracic and substernal tumor masses revealed similar basic structures with minor differences apparent in the sections. All showed very cellular areas of embryonal connective tissue alternating with areas of immature cartilage with a pale-staining matrix.

The embryonal connective tissue cells were pleomorphic, varied in shape from oval to spindle and elongated forms and contained pink cytoplasm and large, dark nuclei. There was a loose intercellular substance containing many fine, pink fibrillar elements. A zone of transition occurred between this tissue and the cartilage in which the cells became rounded and were surrounded by a deep pinkish-staining matrix. The lacunae were irregularly arranged in relation to one another, and the cells in the lacunae were similar to those previously described.

Deep blue-staining masses indicating deposits of calcium were seen in the thoracic tumors. Irregular trabeculae of bone arose in the midst of the cartilage. In the substernal masses there was a predominance of the cellular (fibroblastic) portion of the tumor. In all the sections the tumor had invaded and separated the adjoining skeletal muscles.

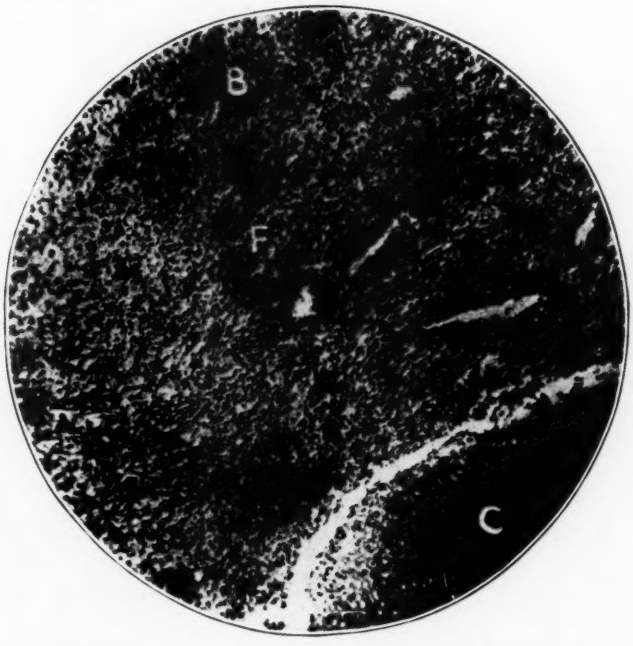

Fig. 4.—Section of the tumor of the left orbit. *F* indicates fibroblastic tissue; *C*, immature cartilage, and *B*, bone spicule.

In structure the orbital tumor was quite similar to the tumor masses described. Zones of exceedingly cellular, embryonal connective tissue, in which there were many mitotic figures, alternated with areas of pale and deeper pinkish-staining cartilage. Here and there spicules of fibrous bone were evident by their more compact structure and blue stain. There were few blood vessels.

Sections of the posterior portion of the intra-orbital section of the optic nerve showed numerous cystic spaces in the nerve bundles. The intervening portions of the nerve were finely honeycombed, but the medullary sheaths appeared to be intact. Such abnormalities were not noted in the anterior section of the nerve (up to 10 mm. behind the sclera), the structure appearing quite normal.

No evidences of a new growth or other pathologic changes were found in the posterior segment of the globe.

COMMENT

Sarcoma in an orbital bone in the presence of a similar growth in other parts of the body is unique. This case also differs from the few reported intances of metastatic orbital sarcomas in that it is not possible to point with accuracy to the primary source of the new growth. Except for the invasion of adjacent muscle tissues by the tumor, the growth was confined to separate skeletal bones. Although the first symptoms pointed to a primary growth in the dorsal spine, one cannot with certainty rule out the possibility of a more or less simultaneous involvement of the bones involved, i.e., multiple sarcomatosis. Such a diagnosis is supported by the embryonal character of the cellular and fibrous tissues in all the masses studied and by the presence of immature cartilage and bone. It appears that undifferentiated tissues in these various areas suddenly received a stimulus to grow and assumed the proportions of malignant tumors.

VISUAL FIELD DEFECTS IN PREGNANCY

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INTRODUCTION

In 1922, I read before the International Congress of Ophthalmology, held at Washington, a paper entitled "Bitemporal Contraction of the Visual Field in Pregnancy," with a report of the findings in the visual field in thirty-one cases of normal pregnancy with no ocular lesions, in twenty-two (70.97 per cent) of which there were changes in the limits of the visual field of the nature of bitemporal contraction, which I associated with the normal enlargement of the hypophysis in pregnancy, with a corresponding compression of the chiasm.

In this paper I referred to the fact that similar defects had been reported by Bellinzona and Tridontani [1] in 1903, and by Forti [2] in 1910. A subsequent review of the literature by others and myself showed that Igersheimer,[3] in 1916, in an article on the detection of morbid processes in the visual pathways, referred to deformation of the visual field in the nature of a bitemporal defect as an almost constant phenomenon in pregnancy, reporting its occurrence in twelve cases. Other isolated cases had also been reported.[4] My paper, however, was the first in which detailed figures of the findings in the visual field in this condition were reported for a series of cases.

In the discussion of my paper, Dr. Lancaster and Dr. Cavill [5] presented a detailed account of their findings in sixty cases from the maternity department of the New England Hospital for Women and Children. They reported positive results in 80 per cent of the cases, all contractions over 5 degrees being considered as such.

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1. Bellinzona and Tridontani: Boll. d. Soc. med.-chir. di Pavia **35**:37, 1904.
2. Forti: Arch. di ottal. **17**:357, 1910.
3. Igersheimer: Heildelb. Ber., 1916, p. 352.
4. Thompson, quoted by Abramovicz.[6d] Reus: Wien. klin. Wchnschr. **21**:1175, 1908.
5. Lancaster and Cavill: Tr. Internat. Cong. Ophth., 1922.

Subsequently several investigators [6] published confirmative findings; others,[7] however, did not obtain such results. Among the former, some attributed the occurrence of the defect to the hypophyseal enlargement of pregnancy, others to toxic influence due to endocrine disturbance or some other disorder, to a general reduction of retinal vitality due to endocrine insufficiency, to suggestive influences or to other causes independent of the hypophyseal hypertrophy, which they did not consider capable of producing this defect by pure mechanical action.

With the opportunities at my disposal at the Ophthalmic Institute of the Presbyterian Hospital, with the splendid cooperation obtainable from the different departments of the College of Physicians and Surgeons and with the advantages of the experience furnished by prior examinations and the more accurate modern equipment available and the improvements in technic to date, I again took up the study of the subject, with the purpose of clearing up some of the moot points. I report here the results of my first findings in one hundred and eight cases.

MATERIAL AND METHODS

All the patients were from the antepartum department of the Vanderbilt Clinic, and their visual fields were taken with the Feree-Rand perimeter according to the recommended technic, with an illumination of 7 candle power.[8] Only patients with no general complications and presenting no ocular lesions were studied. They were examined mostly with 5/330 and 1/330 white tests and 5/330 red and blue tests, the last being utilized especially in cases with normal findings for the white tests. Great care was taken to avoid anything in the nature of suggestion, the patient having no indication of what was expected in relation to her answers. Fatigue on the part of the patient was also avoided. Of course, not all the patients were equally intelligent or responsive, and care and attention were required in making the preliminary explanation and in watching the patient during the examination to prevent any deviation of the eye from the fixation object during the tests. With one exception, all the patients were in the last three months of pregnancy. Several were examined on more than one occasion prior to their confinement, and in some cases postpartum examination was also made one or more times.

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6. (*a*) de Schweinitz, G. E.: Tr. Ophth. Soc. U. Kingdom **43**:12, 1922. (*b*) Ashikaga: Klin. Monatsbl. f. Augenh. **73**:554, 1924. (*c*) Löhlein, W.: Monatschr. f. Geburtsh. u. Gynäk. **65**:129, 1925. (*d*) Abramovicz: Brit. J. Ophth. **11**:17, 1927. (*e*) Johns, J. P.: Am. J. Ophth. **13**:956, 1930.

7. (*a*) Rowe, Bank and Alcott: Am. J. Physiol. **80**:660, 1925. (*b*) Shoningen: Klin. Monatsbl. f. Augenh. **73**:11, 1926. (*c*) Metzger, E. L.; Simon, M., and Weinberg, H.: Monatschr. f. Geburtsh. u. Gynäk. **70**:140, 1925. (*d*) Richter: Klin. Monatsbl. f. Augenh. **75**:475, 1925.

8. In a comparative study of perimeter findings it is always necessary to use the same instrument and to specify the instrument used. In a few comparative tests with the 1/330 white test with the Feree-Rand and the Lister perimeter, I found that the same patient almost always showed a larger field with the latter, possibly because of the greater contrast of the black background, making it easier to determine when the small test object came into the field.

In the determination of the normal or abnormal character of the individual fields, the Traquair standards were adopted,[9] with a 10 degree margin, the 5 degree margin hitherto used by most observers being considered too small. I classified the results as positive, negative (normal) and doubtful, the last classification including borderline cases.

The tests used in these examinations and the number of cases in which they were carried out are shown in table 1.

TABLE 1.—*Tests and Number of Cases*

	Number of Cases
12/330, 5/330 and 1/330 white tests	1
5/330 white test	14
5/330, 3/330, 2/330 and 1/330 white tests and 5/330 color test	2
5/330, 2/330 and 1/330 white tests	1
5/330 and 2/330 white tests	1
5/330 and 3/330 white tests	1
5/330, 2/330 and 1/330 white tests	1
5/330 and 2/330 white tests	1
5/330 and 1/330 white tests	48
5/330 and 1/330 white tests and 5/330 color test	5
1/330 white test	16
1/330 white test and 5/330 color test	17
Total	108

A summary of these statistics is as follows:

Examination with 5/330 white test	77 cases
Examination with 1/330 white test	76 cases
Examination with 3/330 and 2/330 white tests	7 cases
Examination with 5/330 color test	24 cases

FINDINGS

The results of these examinations were:

Positive findings in	73 cases	(67.59%)
Negative findings in	23 cases	(21.29%)
Doubtful findings in	12 cases	(11.11%)

In the study of the character of the defect, I took the seventy-three patients of the first group into consideration. Of these, ten were examined with the 5/330 white test, forty-seven with the 5/330 and 1/330 white tests, five with the 5/330 and 1/330 white tests and the 5/330 colored test, one with the 1/330 white test and ten with the 1/330 white test and the 5/330 color test. In fifteen, defects were found with the 1/330 white test which did not show with the 5/330 white test; in two, the defect appeared with the color tests, the 5/330 and 1/330 white tests giving negative results. In eleven other cases the defect was observable with the color test, the 1/330 white test giving negative results. The defect was found in both eyes in fifty-five cases; it was limited to the right eye in eight and to the left eye in ten cases.

9. Traquair, H. M.: Clinical Perimetry.

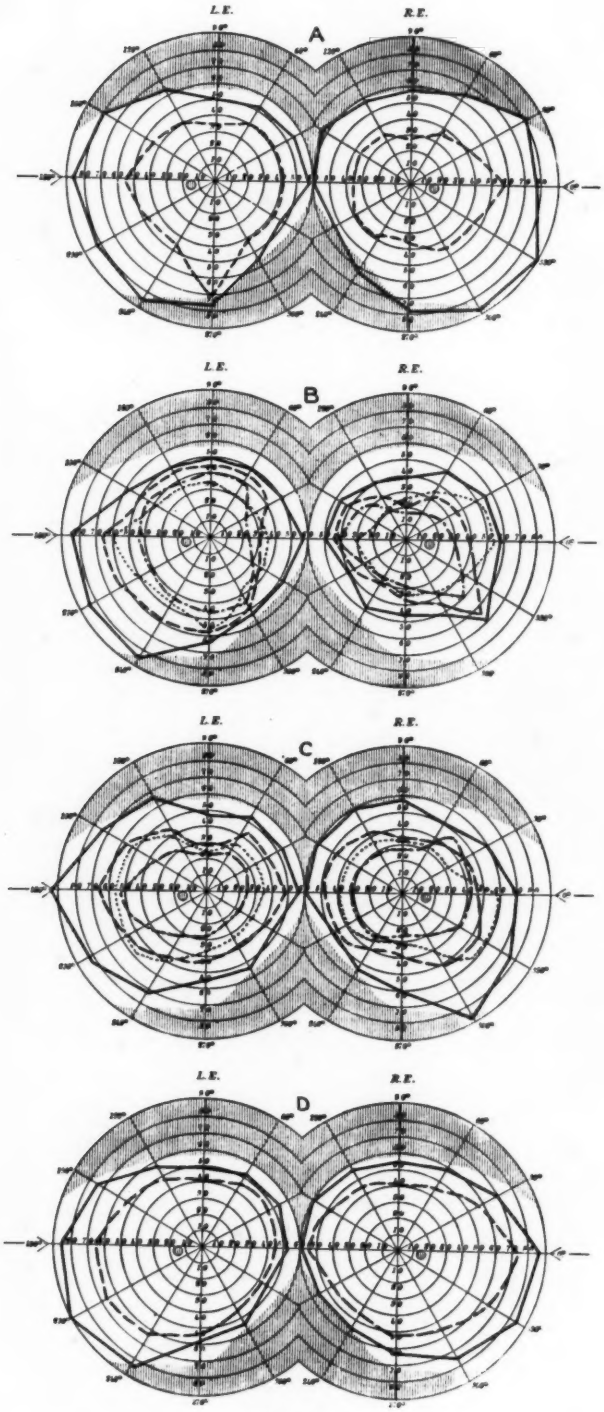

Figure 1

EXPLANATION OF FIGURE 1

Fig. 1.—*A* (case 5), visual fields at antepartum examination, on Jan. 18, 1933, showing diffuse temporal contraction with the 1/330 white test (line with long dashes), with a normal field for the 5/330 white test (unbroken line). *B* (case 8), visual fields at antepartum examination, on Dec. 27, 1932, showing marked diffuse temporal contraction, which is more pronounced in the right eye, especially in the upper and outer quadrant, with the 2/330 white test (line with long and short dashes) and the 3/330 white test (line with long dashes) and in the upper and outer quadrant of the left eye with the 10/330 red test (line with short dashes). As in *A*, the unbroken line indicates the 5/330 white test. *C* (case 30), visual fields at antepartum examination, on Feb. 7, 1933. In the right eye the field shows diffuse temporal contraction with the 5/330 white test (unbroken line), the 1/330 white test (line with long dashes), the 5/330 red test (line with long and short dashes) and the 5/330 blue test (dotted line), with a more pronounced contraction of the quadrant with the blue test. In the left eye the field shows diffuse temporal contraction with the 1/330 white test and the 5/330 blue test, with a dip above and contraction of the quadrant with the 5/330 red test. *D*, visual fields of same patient as in *C*, at postpartum examination, on June 5, 1933; the field of the right eye is normal, and that of the left eye is almost normal. In all the figures *L* indicates the left eye and *B* the right.

The deformation of the visual field was mostly found to be in the nature of a temporal contraction; it was not necessarily limited to the upper and outer quadrant. A limitation to this quadrant or a more pronounced defect in this direction was found in thirty-one cases. Concentric limitation was noted in two cases.

Some of these defects may be noted in the accompanying charts. Those belonging to case 5 (fig. 1 *A*) show general temporal contraction with the 1/330 white test; those of case 8 (fig. 1 *B*) show diffuse temporal contraction with all the tests, which is more pronounced in the upper and outer quadrant with the 2/330 and 3/330 white tests in the right eye and with the 10/330 red test in the left. In case 30 (fig. 1 *C*) on antepartum examination there was diffuse temporal contraction with the 5/330 and 1/330 white tests and the 5/330 red test, with a contraction of the quadrant for blue in the right eye and for red in the left, there being also some tendency to concentric contraction for these colors. Postpartum examination (fig. 1 *D*) in this case showed almost complete recovery of the normal limits of the field. Antepartum examination in case 35 (fig. 2 *A*) showed normal limits of the field with the 5/330 and 1/330 white tests, with concentric contraction for red and blue. In case 43 the fields showed diffuse temporal contraction on antepartum examination (fig. 2 *B*), which was more marked in the upper and outer quadrant, and almost normal fields on postpartum examination (fig. 2 *C*). In case 59 (fig. 3 *A*) the fields showed diffuse temporal contraction on antepartum examination. Postpartum examination in the same case (fig. 3 *B*) revealed normal fields. The fields in case 52 (fig. 3 *C*) showed diffuse temporal contraction, which was more marked in the upper and outer quadrant. Those in case 71 (fig. 4 *A*) showed moderate diffuse temporal contraction on antepartum examination; postpartum examinations in the same case (4 *B*, *C* and *D*) revealed an increase of the defect, with a slight final improvement. In forty of the cases several antepartum examinations were made; an increase of the defect was noted in the later examinations in sixteen, a decrease in twelve.

On postpartum examination, which was made in twenty-six cases, a more or less pronounced decrease or abolition of the defect was noticeable in twenty (figs. 1 *D*, 2 *C* and 3 *B*). An increase was noted in five; this was especially pronounced in case 71 (fig. 4 *B*, *C* and *D*), in which it was somewhat suggestive of hypophyseal growth stimulated by pregnancy, such as has been reported by Shimkin [10] and others.[11] I saw a

10. Shimkin, N.: Folia ophth. orient. **1**:90, 1932.

11. (*a*) Jung: Klin. Monatsbl. f. Augenh. **81**:578, 1922. (*b*) Gross: ibid. **2**:802, 1924. (*c*) Patry, A.: Ann. d'ocul **167**:14, 1930. (*d*) Fischer: J. Anat. (Nov.) 1931. (*e*) Rauh, W.: Klin Monatsbl. f. Augenh. **138**:249, 1931.

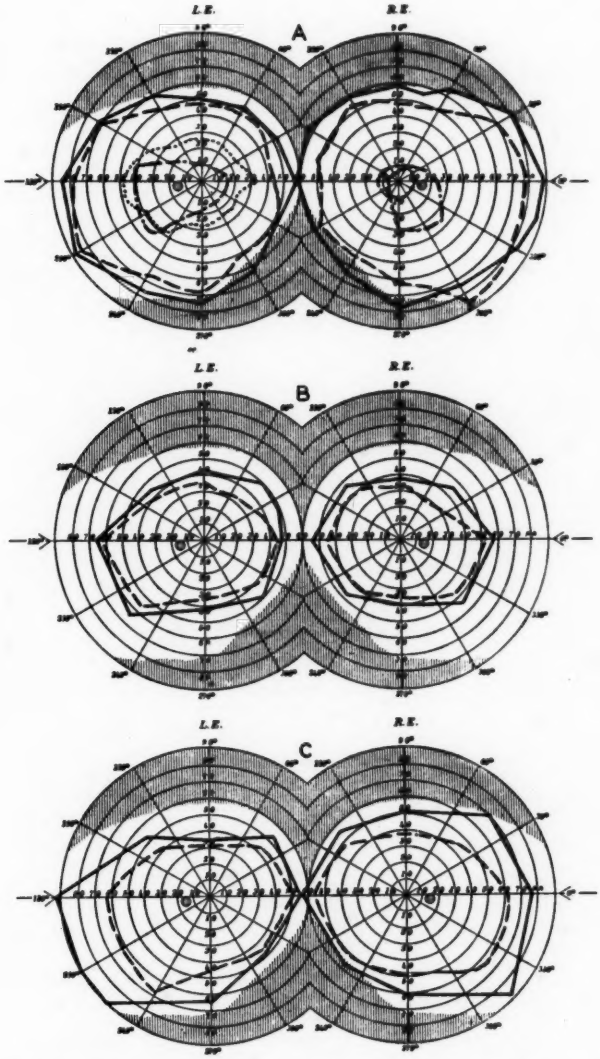

Fig. 2.—*A* (case 35), visual fields at antepartum examination, on Feb. 17, 1933. These were normal with the 5/330 (unbroken line) and 1/330 (line with long dashes) white tests, but showed concentric contraction with the 5/330 red test (line with long and short dashes) and the 5/330 blue test (line with short dashes), which was more marked in the right eye. *B* (case 43), visual fields at antepartum examination, on May 2, 1933, showing diffuse temporal contraction, which is more marked in the upper and outer quadrant. The unbroken line indicates the 5/330 test; the line with long dashes, the 1/330 test. *C*, visual fields of same patient as in *B*, at postpartum examination, on Aug. 2. These were almost normal. The lines have the same significance as in *B*.

TABLE 2.—*Frequency of Findings in Relation to Duration of Pregnancy*

Stage of Pregnancy	Findings			Total	Percentage of Positive Findings
	Positive	Negative	Doubtful		
4 months	0	1	0	1	0
7 months	12	5	1	18	83.33
8 months	27	8	5	40	67.50
9 months	34	9	6	49	70.83
Total	73	23	12	108	67.59

TABLE 3.—*Frequency of Findings in Relation to Number of Pregnancies*

Number of Pregnancies	Findings			Total	Percentage of Positive Findings
	Positive	Negative	Doubtful		
1	42	14	7	63	66.66
2	15	6	5	26	57.69
3	10	2	0	12	83.33
4	2	1	0	3	66.66
5	2	0	0	2	100.00
7	1	0	0	1	100.00
8	1	0	0	1	100.00
Total	73	23	12	108	67.59
1 and 2	57	20	12	89	64.04
More than 2	16	3	0	19	84.21

TABLE 4.—*Frequency of Findings in Relation to Age*

Age, Years	Findings			Total	Percentage of Positive Findings
	Positive	Negative	Doubtful		
17	1	0	0	1	100.00
18	3	0	0	3	100.00
19	0	2	0	2	0
20	5	1	0	6	83.33
21	6	5	1	12	50.00
22	7	1	4	12	58.33
23	10	2	0	12	82.33
24	4	1	0	5	80.00
25	7	3	1	11	63.63
26	6	2	1	9	66.66
27	1	0	1	2	50.00
28	4	1	0	5	80.00
29	4	1	1	6	66.66
30	3	2	2	7	42.85
31	0	0	1	1	0
32	1	1	0	2	50.00
33	3	1	0	4	75.00
35	2	0	0	2	100.00
36	2	0	0	2	100.00
37	2	0	0	2	100.00
40	1	0	0	1	100.00
41	1	0	0	1	100.00
Total	73	23	12	108	67.59
From 17 to 30 years	61	21	11	93	64.94
From 31 to 41 years	12	2	1	15	80.00

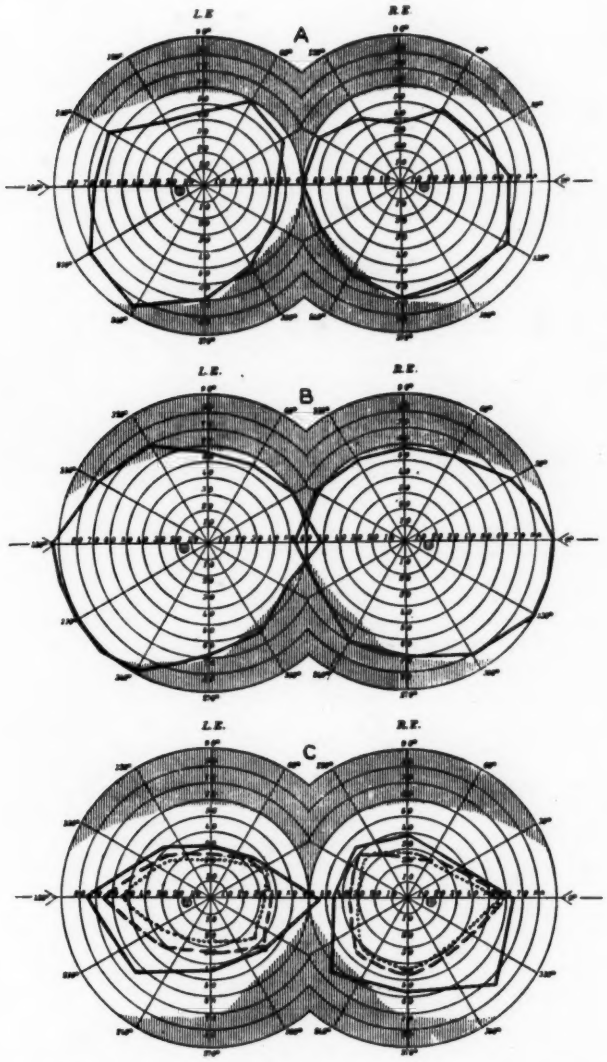

Fig. 3.—*A* (case 59), visual fields at antepartum examination, on March 22, 1933, showing diffuse temporal contraction with the 5/330 test (unbroken line). *B*, visual fields of same patient as in *A*, at postpartum examination, on May 16; these were normal with the 1/330 white test (unbroken line), the 5/330 blue test (dotted line) and the 5/330 red test (line with long dashes). *C* (case 52), visual fields at antepartum examination, on June 22, 1933, showing diffuse temporal contraction, which was more marked in the upper and outer quadrant with the 5/330 white test (unbroken line).

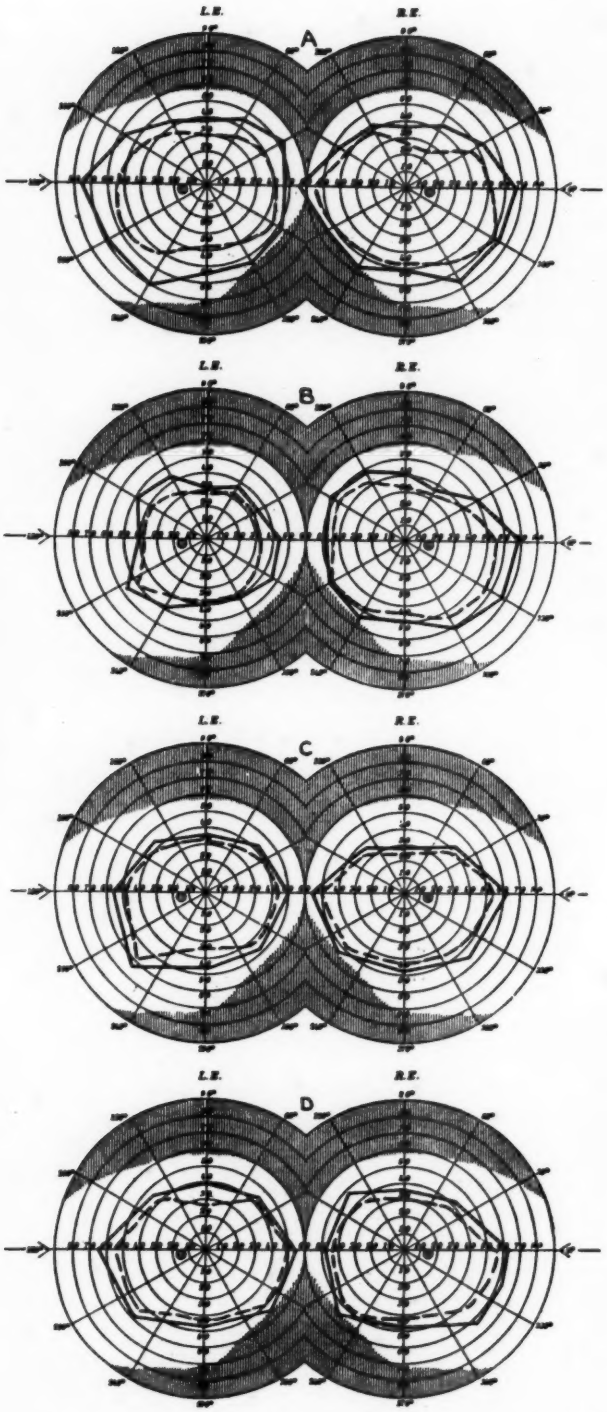

Fig. 4 (case 71).—*A*, visual fields at antepartum examination, on April 4, 1933, showing diffuse temporal contraction with the 5/330 white test (unbroken line) and the 1/330 white test (line with long dashes). *B*, *C* and *D*, visual fields at postpartum examination on May 31, June 14 and August 5, respectively, showing an increase, with some improvement at the final examinations.

similar case in the daughter of a Cuban *confrère,* which I have not reported. In another of the cases studied a similar but not too pronounced increase of the defect was noted at first examination, but some improvement took place subsequently. In the other cases the increase was slight.

The relation of these defects to the stage of pregnancy can be noted in table 2. This shows a slight increase in the ninth month. The increase observable in the seventh month cannot be taken into account, owing to the small number of cases of this group.

The relation of the defects found to the number of pregnancies is shown in table 3. A marked increase in frequency is noted after the second pregnancy.

TABLE 5.—*Frequency of Findings in Relation to Age in Primiparae Only*

Age, Years	Findings			Total	Percentage of Positive Findings
	Positive	Negative	Doubtful		
17	1	0	0	1	100.00
18	2	0	0	2	100.00
19	0	1	0	0	0
20	3	1	0	4	75.00
21	4	4	1	9	44.44
22	5	0	1	6	83.33
23	6	1	0	7	85.71
24	2	0	0	2	100.00
25	3	1	0	4	75.00
26	4	2	1	7	57.14
27	4	0	1	5	80.00
28	1	1	0	2	50.00
29	3	1	1	5	60.00
30	1	2	2	5	20.00
35	1	0	0	1	100.00
37	1	0	0	1	100.00
Total	41	14	7	62	66.12
From 17 to 35 years	26	8	2	36	72.22
From 25 to 37 years	15	6	5	26	57.69

The influence of age was studied first, without any discrimination (table 4) and, second, taking only primiparae into consideration (table 5). In the first group there was a marked tendency for the defect to occur after the age of 30, but in the primiparae the proportion was reversed.

As regards the influence of race, the defect seemed to be relatively more frequent in the colored race and Latin races than among the Nordics.

COMMENT

From a study of these findings, there is no question but that, in a majority of cases of normal pregnancy, defects of the visual field occur. These are of the nature of bitemporal contraction, with some tendency to limitation to a quadrant or intensification in a quadrant. Except in some of the tests with colors, there was no tendency to concentric contraction in the cases studied. This contraction was more marked and

more noticeable in tests with small objects (1/330). It was more apparent in the last months of pregnancy and was more marked after successive pregnancies. Age did not seem to have any influence on its production. A greater predisposition for this defect seems to exist in the colored race and the Latin races than among the Nordics.

In regard to the production of this defect, one must consider especially the influence exercisable by the normal hypertrophy of the hypophysis in pregnancy, to which Compte [12] first called attention and which was accurately determined by Erdheim and Stumme,[13] this hypertrophy reaching to from two to three times the original volume.

The anatomic relations of the hypophysis with the chiasm, which were accurately determined by Schaeffer and which were described by de Schweinitz in his classic Bowman Lecture of 1923, vary considerably as to the position of the chiasm in the opening of the sella turcica, the distance separating the under surface of the chiasm from the upper surface of the hypophysis (from contact to 10 mm.) and the density of the diaphragma sellae separating both structures.

In connection with the effects of pressure produced by growths in the chiasm, it is well to consider Traquair's [9] views as to the mechanism of the production of defects:

> There are three hypotheses: pressure, traction and toxins. The first is the most attractive, if it is understood that pressure acts not only directly on the nerve fibers, but also on the blood vessels, impeding the venous return with production of arterial ischemia.

It is possible that the normally hypertrophied hypophysis, even when not directly compressing the chiasm, may produce vascular changes of the nature of arterial or venous engorgement or anemia in or around the chiasm. These may be accompanied by more or less secondary edema, with secondary changes in the tissues of the nerve fibers affecting their conductivity, such as Foster Kennedy has described as "pressure neuritis" in relation to tumors of the frontal lobe.

It would have been desirable to have had the opportunity to examine the anatomic relations at autopsy in some of the patients who died at the end of the pregnancy, but in the only case in which death occurred during the period of research, at the Sloane Hospital for Women, permission to open the cranium was refused.

The character of the contraction seems to preclude any toxic element in its production, except, perhaps, in connection with the color defects of the visual field.

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12. Compte: Thèse de Lausanne, 1898.

13. Erdheim and Stumme: Beitr. z. path. Anat. u. z. allg. Path. **46**:132, 1909.

I do not see how suggestion can have any influence, if care is taken not to have the patient know what is expected.

Complementary examinations, such as examinations with the Walker screen with a 1/2,000 test, and examinations of the visual field in non-pregnant women treated with an anterior pituitary-like principle from the urine of pregnant women have been undertaken to clear up some of the obscure points. It is desired to carry out examinations at autopsies in suitable cases.

SOME PRINCIPLES OF PLASTIC SURGERY OF THE EYELID, WITH SPECIAL REFERENCE TO THE HUNGARIAN SCHOOL

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CHICAGO

According to Hirschberg,[1] the historian of ophthalmology, the ancient Greeks and Romans were not acquainted with plastic surgery. There is no mention of plastic surgery of the eyelids in the scanty discussions concerning surgery which remain from the Greeks. Celsus, a Roman, who lived from 25 B. C. to 50 A. D., wrote extensively on the subject of surgery in his encyclopedia. The part pertaining to medical science was a free translation from the Greek, not without errors and superficial additions. In this he wrote that a small quadrilateral defect could be covered by a sliding quadrilateral flap which had been undermined on three sides (fig. 1 *a*). He recommended the use of this procedure for defects of the ears, lips and nose. This is the ***methode par glissement,*** which the French claim as their own. Although Celsus has been credited with having devised many operative procedures, at no time did he claim a procedure as his own, and Hirschberg wrote that Celsus did not devise anything.

Hirschberg claimed that the true originators of plastic surgery were, without doubt, the Indians. Necessity prompted the devices. At the dawn of history in India the nose was removed as a punishment for crime and infidelity, and for purposes of vengeance in mutilation of prisoners of war. In the old "Canon of the Indian Healing Art" it is commanded as follows:

> If someone has his nose cut off, the surgeon must cut off from a tree a leaf of similar size, place it upon the cheek and cut out from it a piece of skin and flesh of the same size, but in such a manner that it is still attached at one place, suture the defect in the cheek with needle and thread, refresh the cut surfaces of the nose, turn up over it, quickly but carefully, the excised skin, attach it well, suture the new nose securely and cover it with a well fitting bandage.

Hirschberg stated that plastic repair of the nose was first done in Europe at the beginning of the fifteenth century. The operation was

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1. Hirschberg, J.: (*a*) Geschichte der Augenheilkunde, in Graefe-Saemisch: Handbuch der Augenheilkunde, ed. 2, Leipzig, Wilhelm Engelmann, 1899, vol. 12, p. 241; (*b*) ibid., ed. 2, 1911, vol. 14, pt. 2, p. 96.

performed by the Sicilian surgeon, Branca. It is not improbable that he received advice and suggestion in some unknown manner from India, for he also repaired the nose with tissue secured from the cheek. Branca's son, Antonium, however, devised the procedure whereby the nose was built of skin from the arm which had been fastened to the head. This is known as the Italian procedure and is still employed by some surgeons for plastic repair of the eyelids.

When and by whom the choice of the forehead instead of the cheek as the source of the flap was introduced is not known. But the taking of a pedicled flap from the forehead is only an alteration of the procedure which is designated as the Indian method. The Fricke method for

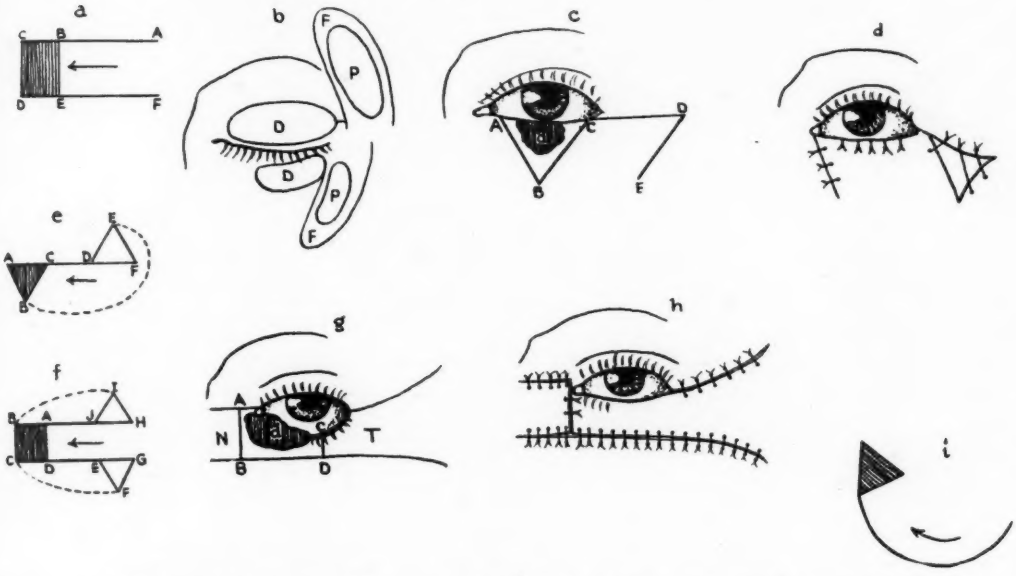

Fig. 1.—(*a*) The quadrilateral defect *BCDE* is covered by the flap *ABEF*, which is stretched and under tension. (*b*) The defects *D* are covered by pedicled flaps *F*, which are secured from the forehead or the cheek and are larger than the patterns *P* of the defects. (*c*) The triangular defect produced following the excision of the tumor mass *d* is covered by the pedicled flap *BCDE*. (*d*) Method of repairing triangular defect produced by the procedure shown in *c*. (*e*) The dotted line shows the approximate extent of the undermined skin, which is displaced in the direction of the arrow. The defect *ABC* is eliminated; *BC* is sutured to *AB*, and the Burow triangle *DEF* is closed by sutures so that *EF* is approximated to *DE*. (*f*) The defect *ABCD* is covered by the flap *HADG*, without tension, by the use of the Burow triangles *EFG* and *HIJ;* the dotted lines bound the area of skin which is undermined. (*g*) Tissue in the area of the defect *d* is so removed that a quadrilateral defect *ABDC* results; this is repaired by union of the nasal *N* and the temporal *T* flaps. (*h*) Sutures used to join flaps in the procedure shown in *g*. (*i*) Method of covering a triangular defect by shifting a flap in the form of half a circle.

the repair of defects in either the upper or the lower eyelid is merely a utilization of these procedures.

In a method described much later, the flap was without a pedicle and was secured from a distant part of the body of the patient. In 1817, there appeared in the *Gazette de Santé* a description by a general of the excision of his nose and of its successful repair by a Hindu, who used a stalkless flap from the buttock.

Fricke,[2] in 1829, wrote concerning plastic surgery of the eyelid. He described a procedure the principle of which was to cover an egg-shaped defect of the skin of the eyelid by a tongue-shaped flap from the neighborhood (fig. 1 *b*).

The real founder of plastic surgery was Dieffenbach.[3] In 1835, he devised a sliding flap for plastic repair of the eyelid. In this procedure the pathologic area is excised in the shape of a triangle. The skin adjacent to the defect is dissected in the shape of a rhombus with an attached broad lower bridge, drawn over to the defect and affixed and sutured above, as well as on the nasal side (fig. 1 *c*). The quadrilateral defect produced is converted into a smaller and triangular defect by obliquely placed sutures (fig. 1 *d*). By means of cicatrization of the remaining temporal defect, the implanted flap is stretched and made smooth.

Many modifications of the principles of Dieffenbach and Fricke have been described and employed.

Burow,[4] in the first half of the nineteenth century, described a plastic procedure which consisted in excision of a triangular area at the end of the incision. This allowed the flap to slide more easily in the desired direction. Burow practiced this method with a straight line incision. He excised one peripheral triangle for the purpose of covering a triangular defect (fig. 1 *e*) and two peripheral triangles for the purpose of covering a quadrilateral defect (fig. 1 *f*).

The methods credited to Celsus and Burow are the foundation for a number of new modifications. One recognizes these methods in Knapp's [5] procedure for restoration of a portion of the lower eyelid. The procedure was suggested to Knapp by a student of his at the University of Heidelberg after a case had been presented and discussed by him in class. The pathologic area is removed by straight line incisions so that a quadrilateral defect is produced (fig. 1 *g*). An incision

2. Fricke: Bildung neuer Augenlider (Blepharoplastik) nach Zerstörung und dadurch hervorgebrachter Auswärtswendung derselben, Hamburg, 1829; cited by Elschnig, A.: Handbuch der Augenheikunde, ed. 2 and 3, Berlin, Julius Springer, 1922, p. 373.

3. Dieffenbach: Einige Bemerkungen aus und über Paris, Wchnschr. f. d. ges. Heilk. **3**:7, 1835.

4. Burow, A., cited by Hirschberg.[1b]

5. Knapp, H.: Arch. f. Ophth. **13**:183, 1867.

is made in the outer canthus and is prolonged in a slightly curved form toward the temple and up. A second incision is made which continues the lower border of the quadrilateral defect, and its terminal part is directed toward the temple and down. The flap of skin thus outlined is freed. On the nasal side the incisions are prolonged, and this flap is also loosened. The medial boundary lines of the flap *AB* and *CD* (fig. 1 *g*) are then united with sutures (fig. 1 *f*). It is evident that the union of the flaps cannot be accomplished without a great deal of tension. To relieve this tension, a Burow triangle may be removed above and below, from the base of the temporal flap.

It has been stated that Celsus showed that a triangular defect could be covered by shifting a flap which had the form of a half circle (fig. 1 *i*). If one is to believe Hirschberg, Celsus did not devise anything. This procedure, whoever the deviser may have been, was not practiced and rested in oblivion.

CONTRIBUTIONS OF THE HUNGARIAN SCHOOL

The contributions of the Hungarian School to the principles of plastic surgery of the eyelid consist in a restoration of this principle and a combination of various original modifications of it with principles of others, as Burow's triangles. This has produced a method which gives most satisfactory results and which can frequently be used to advantage in plastic surgery of other parts of the body. The Hungarians who have advanced this branch of ophthalmic surgery to its present state are Siklóssy Sr., Blaskovics and Imre Jr.

Siklóssy,[6] in the latter part of the nineteenth century, devised a procedure which gave fairly good results in cases of ectropion of slight or moderate degree. The operation consisted of an incision which began from a point a few millimeters above the inner canthus, halfway between the base of the bridge of the nose and the inner commissure and extended in a straight line downward and somewhat toward the temple (fig. 2 *a*). The skin on the temporal side of the incision was undermined from its substratum toward the margin of the eyelid and was elevated so that *B* was sutured to *F*. The location of *B* depended on the size of the ectropion. The point *C* was sutured to *G*, *D* to *H*, and *E* to *I*. This is the principle of oblique sutures. When this procedure was completed, a fold of skin was present at each end of the incision. These folds were allowed to flatten out, which they did within a few weeks after the operation.

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6. Siklóssy, G.: Zur Blepharoplastik, Arch. f. Augenh. **31**:186, 1895. The description of this procedure was not published, and Siklóssy soon discarded it for the procedure shown in figure 11. This information was given to the author by Professor Blaskovics.

Siklóssy and his pupils practiced this procedure, but the direction of the incision was modified so that it was bow-shaped (fig. 2 *b* and *c*). The area of skin undermined was greater. The length and the curvature of the incision depended on the degree of ectropion. These modifications resulted in the correction of ectropion of greater degree than was possible with the original procedure.

Between 1895 and 1900, Blaskovics [7] improved Siklóssy's procedure by the addition of a Burow triangle at each end of the incision. The apex of the upper triangle was directed toward the eye, that of the lower one toward the nose. This allowed the flap to slide more easily and more completely and did away with the folds at each end of the incision.

In time Blaskovics altered the direction of the incision. About 1912 he began the incision 1 cm. to the nasal side of the inner canthus and

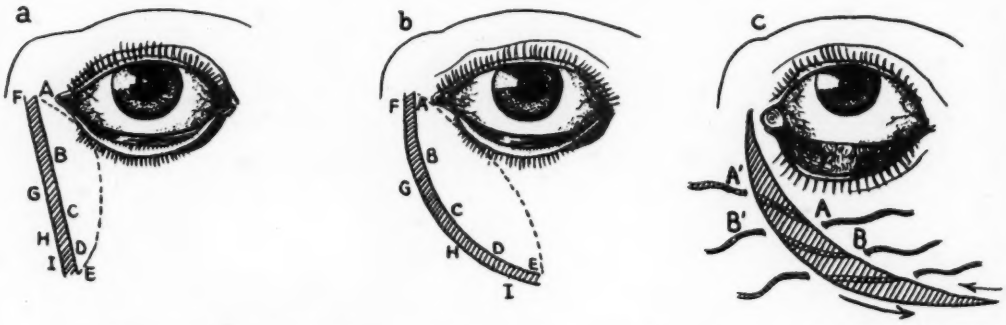

Fig. 2.—(*a*) Siklóssy's procedure, with a straight, downward incision and oblique sutures; *B* is sutured to *F*, *C* to *G*, *D* to *H* and *E* to *I*. (*b*) Length and curvature of incision for the repair of ectropion of moderate degree (Siklóssy's procedure). (*c*) Length and curvature of incision for the repair of ectropion of marked degree (Siklóssy's procedure).

0.5 cm. above the canthal ligament. It was so directed that the upper portion was almost vertical and the lower portion was so curved that it ended at a point below, and in line with, the outer canthus. The continuation of the incision from this point formed the base of the Burow triangle (fig. 3 *a*).

Further changes in the direction of the incision were made by Blaskovics. These pertained mainly to the lower half of the incision just described, which was made less curved, so that the entire incision was only slightly bow-shaped (fig. 3 *b*).

Siklóssy's original procedure and its modifications by himself and Blaskovics were used exclusively for ectropion. It is never used now in such cases. Blaskovics and his followers have used and do use Blaskovics' procedure successfully in the repair of many of the defects

7. Blaskovics: Personal communication to the author.

of the eyelids which result most frequently from the excision of neoplasms.

To Imre Jr.[8a] belongs the credit for having first practiced this procedure, modified and improved by principles which were original with him, for the plastic repair of various defects of the eyelid. An address on this subject was delivered by him before the Royal Hungarian Ophthalmological Society and his writings on this subject appeared at about the same time that the writings of Blaskovics were first published.

When practicable, Imre excised the lesion of the eyelid so that the defect produced was triangular. The incision for the flap began along the base of the triangle and was slightly bow-shaped (fig. 4 *a*). A Burow triangle was excised at the end of the incision. The skin was undermined extensively over an area extending beyond an imaginary line connecting the apex of the Burow triangle and the apex of the defect. The defect produced by the Burow triangle was closed by

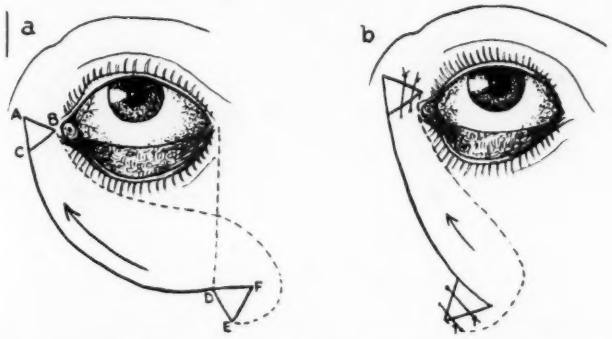

Fig. 3.—(*a*) Blaskovics' modification of Siklóssy's procedure; the incision ends at a point *D* beneath the external canthus. The Burow triangles *ABC* and *DEF* are removed from the ends of the incision. (*b*) Modification by Blaskovics of the incision in *a*, resulting in a less marked curve in the lower half.

sutures. The flap was displaced readily along the line of incision; oblique suturing was used, and the defect was covered without tension.

In time Imre altered the curvature of the incision so that it became more bow-shaped. This is especially interesting in view of the gradual decrease in the curvature of the incision used and recommended by Blaskovics during this same period. In general, Imre now believes that the narrower the defect the greater should be the radius of the incision. In circular defects (fig. 4 *b*) the incision used by him is so curved that the width of the flap equals the diameter of the defect, and the incision terminates almost on the same plane as the upper limits of the defect.

8. Imre, J. Jr.: (*a*) Papers read before the Medical Society, Budapest, 1912, 1913 and 1914 and before the meeting of the Royal Society of Physicians, Budapest, Jan. 17, 1914 (Orvosi hetil., March 15, 1914, p. 227); (*b*) Lidplastik und plastische Operationen anderer Weichteile des Gesichts, Budapest, "Studium," 1928.

Two other modifications originated by Imre are important. He found that by undermining the skin on the side of the incision opposite that of the flap, but leaving the upper side of the Burow triangle adherent to the underlying tissue, he was able to readily cover large defects. This principle is well illustrated in figure 4 *b*. The flap is displaced so as to cover the defect, and the undermined skin is readily approximated to the flap by sliding it in the opposite direction and upward. This follows naturally when the Burow triangle is sutured. The flap is then sutured in place by obliquely directed sutures and covers the defect without tension.

The other modification, which is the more recent one, pertains to the shape of the Burow triangle (fig. 4 *c*). Instead of an equilateral triangle, the apex is rounded and the side of the triangle nearest the

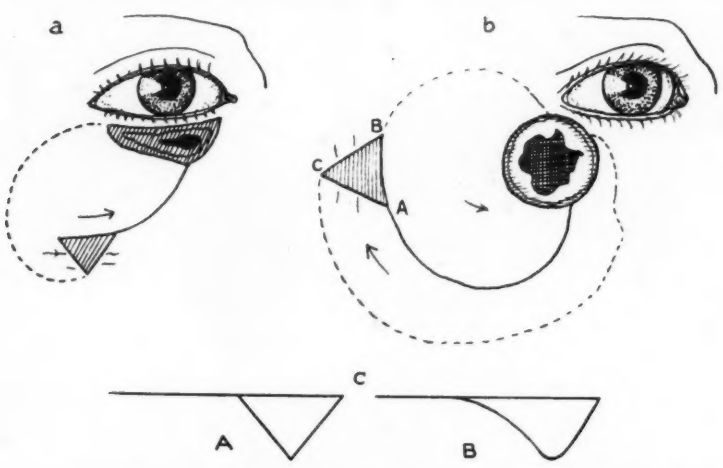

Fig. 4.—(*a*) Imre's procedure in one of his early cases (from his book "Lidplastik"). (*b*) Imre's procedure in one of his more recent cases (from "Lidplastik"). The side of the Burow triangle *BC* remains adherent to its substratum, and *AC* is sutured to *BC;* the skin beneath the incision is undermined extensively. (*c*) *A*, regular Burow triangle; *B*, Imre's modification.

defect curves gracefully over into the line of incision. This allows greater displacement of the flap toward the defect which it is to cover.[9]

It is Imre's contention that Siklóssy's operation, with the modifications devised by his pupils, was not that of a sliding flap but simply an undermining of the skin on one side and its elevation by oblique sutures. This procedure was used principally, if not exclusively, for different degrees of ectropion. A real sliding flap, however, is secured

9. Professor Imre has not as yet described this modification. I saw him employ it and agree with him that it is an improvement on the Burow triangle.

by the curved incision of Imre, together with the undermining of the skin on the side of the incision opposite that of the flap, the use of the Burow triangle or Imre's modification of the Burow triangle, and oblique sutures.

By this method the defect is covered with skin from the immediate neighborhood, without any pedunculated flap. The skin of the immediate neighborhood simulates in color and character that originally present at the site of the defect much more than does that from a distant part, and the flap heals sooner. In order to be able to slide the largest area of skin with the smallest possible wound one must naturally utilize a curved incision and slide the tissue used for covering the defect in a bow. The skin displacement divides itself over a greater terrain the greater the radius of the bow-shaped incision. The curved incision should be about four times as long as the width of the base of the defect. A solution and security for the proper nourishment of the flap is provided by this method.

COMMENT

Naturally, not all defects of the eyelid can be repaired by the method evolved by the Hungarian School. No such claims have ever been made. The principles are found most useful in the plastic repair of defects of the lower eyelid and the inner and the outer canthus and of some of the defects of the upper lid. An idea as to the scope of its usefulness may best be secured by a study of the excellent book on the subject by Imre.[8b]

The Hungarian principles of plastic surgery of the eyelids are to be commended. Critical appraisal, however, reveals that by gradual original alterations of the Siklóssy method for the repair of ectropion a procedure has been evolved which has for its fundamental principle a modification of which was stated some two thousand years ago—a triangular defect can be covered by shifting a flap which has the form of half a circle.

USE OF THE FUNDUS COLORIMETER

LEO L. MAYER, M.D.
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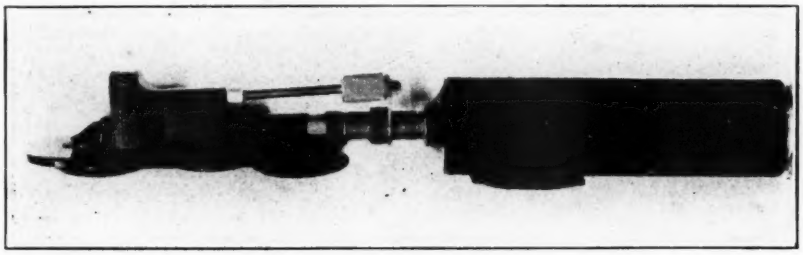
In 1929 Dr. Martin Cohen of New York presented a fundus colorimeter before the American Academy of Ophthalmology and Oto-Laryngology.[1] About two years later, not knowing of Dr. Cohen's instrument, I made contact with an instrument maker of Chicago and was well on the way to producing a colorimeter when in discussing such an instrument with Dr. Cohen at one of the meetings he told me of his fundus colorimeter. I mention all these seemingly nonpertinent details for two reasons: first, because it demonstrates that I had some preconceived ideas on this subject, and secondly, because it explains



The fundus colorimeter.

such a report coming from this source. Dr. Cohen has permitted me to use his original and unique fundus colorimeter for the past twelve months. This report is essentially a summary of the results obtained by the use of this instrument during that period.

DESCRIPTION OF THE COLORIMETER

A circular colorimetric wedge made of gelatin stained with basic fuchsin was constructed under the supervision of Dr. Jones at the Research Laboratory of the Eastman Kodak Company. Dr. Cohen[1] stated: "The wedge has a clear color near its apex, then takes on a faint reddish tint of relatively low saturation, which gradually becomes a deep red color towards its base. It is 18 mm. in diameter, 3 mm. in width, 2 mm. thick, with a hole in the center for mounting, and is sealed with Canada balsam between cover glasses. After the wedge was perfected, it was sent to Mr. Poser of Bausch & Lomb Optical Company, who had it attached with calibration to the hand ophthalmoscope, the instrument thus serving as an electric ophthalmoscope and colorimeter. A narrow, thin, graduated scale surrounds the circular red wedge, reading from zero at the apex of the wedge to

1. Cohen, Martin: Tr. Am. Acad. Ophth., 1929, p. 461.

100 at its base, with division into ten equal parts. The circular wedge with its surrounding scale is attached to the ophthalmoscope and to a rod having at its lower end a wheel that is readily turned, thereby producing a revolution of the combined graduated scale and red wedge. The light from the electric bulb of the ophthalmoscope passes upward through a circular aperture in which the circular wedge and scale revolve. The light then comes in contact with the total reflecting prism of the ophthalmoscope, passes into the patient's eye and is reflected back into the examiner's eye. The light which enters the patient's fundus through the clear apical portion of the wedge appears as a normal whitish disc, similar in size to the normal papilla. This whitish disc is used for fundus examinations. If the color changes in the optic disc are to be studied, the colorimeter is brought into play by gently revolving the wheel which is conveniently located near the handle of the ophthalmoscope. This procedure very gradually changes the color of the whitish projected disc from low to high saturations of red. As soon as the whitish disc on the papilla commences to appear reddish, its reading—which is readily seen on the instrument—is noted; this notation is used for comparison in future examinations of the same case."

In a personal communication, Dr. Cohen recently replied as follows to suggestions made by me:

1. That a constant measurable light source be used.

This is satisfied by checking the standard bulb periodically by means of a photo-electric cell. A small variable resistance unit attached to the base of the instrument may be utilized to give flexibility. The check should be made with the instrument completely assembled. Resistance should be all in circuit when the batteries and bulb are new.

2. That the graduations of wedge now lying between 1 and 40 be extended to the entire circuit.

Although this is probably a valid criticism, if experience shows that settings above 40 are never needed, the expense of procuring a new wedge militates against this change. It was not made.

3. That the wedge be split so that half the field would be white for contrast.

It appeared inadvisable to do this because the red field is already extremely small. Any further reduction would make reading very difficult.

4. That a magnifier and illuminator be supplied for reading the scale in a darkened room.

These may be used as accessories, but to attach them directly to the instrument would render it cumbersome.

5. That a ratchet be provided on the instrument handle in order to lock it in a given position.

This has been provided by a double collar with a small spring between so as to give an even pressure and hold any reading.

USE OF THE INSTRUMENT

Often in certain types of cases the ophthalmologist is asked to estimate the degree of optic atrophy noted in an examination of a fundus, whether the disk is pale and, finally, if there is an increase or a decrease in the atrophy or pallor as seen on a single of many previous examinations. Notwithstanding the fact that many believe that

TABLE 1.—*Values for Normal Persons*

State of Refraction	Number of Cases	Colorimetric Readings	
		Right Eye	Left Eye
Hypermetropia	141	6.6	6.8
Myopia	98	10.9	10.8
Emmetropia	116	7.7	7.8
Average mean		8.4	8.5
Average mean, both eyes		8.43	

TABLE 2.—*Values for Persons with Multiple Sclerosis*

Case	Colorimetric Readings	
	Right Eye	Left Eye
1	8–10	9–10
2	9–12–14	10–14–15
3	16	18
4	10–12–12	9–14–12
5	15–17	22–26
6	26–26–28	20–20–20
Average mean	15.4	16.4
Average mean, both eyes	16.2	

TABLE 3.—*Values for Persons with Miscellaneous Conditions*

Case	Condition	Colorimetric Readings	
		Right Eye	Left Eye
1	Optic atrophy	26–26–26	32–32–32
2	Glaucoma	18–18	10–10
3	Brain tumor	8–6–12–17	15–14–17–17
4	Glaucoma, absolute	35–35	10–8
5	Brain tumor	6–15	6–8
6	Intracranial aneurysm	8–26–26–26	8–6–28–28
7	Paget's disease	11–11–11	12–13–11
8	Retinitis pigmentosa	14–12–12–14	13–14–12–14
9	Brain tumor	31	24
10	Retinitis pigmentosa	16–18–16	15–13–14
11	Brain tumor	8–8–8	8–19–25
12	Chloroma	6–6–25	5–18–31
13	Brain tumor	5–5–19	5–7–8

TABLE 4.—*Values for Persons with Paresis* *

Case	Colorimetric Readings	
	Right Eye	Left Eye
1	10–10–10	10–10–10
2	10–10	8–8
3	9–10–8	10–10–10
4	8–8–8	9–10–6
5	10–9	12–10
6	8–8–10–8	8–8–10–10
7	15	10
8	8–9–10	8–9–9
9	12–14	13–15
10	8–12	8–10
11	10	10
12	9	9
Average	8.8	9.6
Average, both eyes	9.6	

* The fields were normal except in case 7, in which the right eye showed a slight contraction.

a study of the peripheral fields reveals the first evidence of interference with the optic pathways, it was hoped that an evaluation of the color of the disk, if brought to a stage at which minute changes could be

TABLE 5.—*Values for Persons with Syphilitic Optic Atrophy*

Case	Fields	Colorimetric Readings	
		Right Eye	Left Eye
1	Slight contraction	15-14-24-19-18	15-18-18-16-21
2	Slight contraction	19	16
3	Left eye blind	20-20-20-20-20	2)-29-29-29-29
4	No fixation	25	22
5	10 degrees contraction	20-15	20-20
6	Right eye, 10 degrees contraction; left eye, not obtainable	15-16-18-21-21	18-18-24-26-18
7	Normal	20-19	20-20
8	Right eye normal	12-12-12	
9	30 degrees contraction	28	12
10	20 degrees contraction	21	22
11	20 degrees contraction	15-15-15	15-15-20
12	30 degrees contraction	10	10
13	Normal	16	19
14	Right eye, central vision only	10-10	10-10

TABLE 6.—*Values for Persons with Syphilitic Meningitis* *

Case	Colorimetric Readings	
	Right Eye	Left Eye
1	16-16	15-10
2	8-8	8-8
3	8-11	7-9
4	10-10	10-10

* The fields were normal in all cases.

TABLE 7.—*Values for Persons with Tabes* *

Case	Colorimetric Readings	
	Right Eye	Left Eye
1	10-10-10-10	14-14-14-14
2	12-10	14-14
3	12-5-6-8	10-5-8-9
4	10	5
5	8-9	5-11
6	8-8-8	8-8-8
7	8-8-8	8-8-8
8	8	8
9	10-10	14-18
10	12	10
11	8-8-8	
12	8	8
13	8-8	8-8
14	8-8	8-8
15	10-8-10	8-8-10
16	11	12
17	10	10
18	7-7	7-7
19	8-9-8	8-8-8
20	8-10	8-10

* The fields were normal in all cases.

measured, would reveal evidence of extreme value. It is my hope that the fundus colorimeter will fulfil such a need.

During the past twelve months the fundus colorimeter has been used in the examination of a number and variety of persons. Some

were in normal health, others had early stages of neurosyphilis and many showed pallor of the optic nerve head due to a condition such as multiple sclerosis, syphilitic optic atrophy or glaucoma. The tables list the colorimetric readings in the recorded cases. The instrument was used in the Northwestern University Medical School, the Michael Reese Hospital, the Public Health Institute and the Illinois Charitable Eye and Ear Infirmary.

The normal persons are divided into three groups those with hypermetropia, those with myopia and those with emmetropia. So that too many figures may not cloud the issue, only the averages for the normal persons are given. No attempt was made to determine exact refractive errors. In the cases of persons with pathologic conditions frequent examinations of the visual fields were made in an attempt to correlate and compare colorimetric with perimetric findings. The succession of figures indicate the readings on the initial and subsequent examinations. Cases were recorded in sequence unless a complicating condition of the eye made this impossible.

COMMENT

It seems of great importance to repeat the admonitions made by Dr. Cohen, viz., that the same examiner use the instrument, that the amount of illumination should be always the same and that the pupils should be dilated. For normal persons the arbitrarily chosen scale reads between 6 and 11, with a mean of 8.5, the values being lower for persons with hypermetropia and higher for those with myopia. When there is pallor of the disk as a result of lesions in the nerve fibers a range from 12 to 32 has been found. In multiple sclerosis the change is slight at the beginning of the disease, shows a gradual increase and apparently reaches a maximum late in the course, from which maximum there is little change at subsequent examinations. Persons with miscellaneous conditions, such as glaucoma, tumor of the brain, retinitis pigmentosa and idiopathic optic atrophy, show changes in the color of the disk in agreement with our previous conception. Here, too, it seems that a maximum is reached after the disease has progressed for some time, from which point there is no further measurable change. Patients with frank paresis show little if any change in the color of the disks. The visual fields for white and red are, in general, in accord with the foregoing statement. Among the patients with syphilitic optic atrophy there were some whose visual fields were normal or very slightly contracted though the colorimetric readings showed marked atrophy There were other patients who showed marked changes in the visual fields while the colorimetric readings revealed no atrophy or very little. Study of a larger group of such patients may

lead to some conclusion as to the correlation of the changes in the fields and those recorded by the colorimeter. One of the four patients with syphilitic meningitis showed mild atrophy with no changes in the visual fields. In none of the patients with tabes were changes in the visual fields or atrophy of the disks found.

Modifications of the instrument as now constructed are recommended, as follows:

1. That a measurable, constant source of illumination be applied.

2. That the basic fuchsin wedge be so regulated that the portion which now covers the scale from 1 to 40 be spread over the entire scale from 1 to 100.

3. That the wedge be halved in order that the color of the disk with white illumination may be contrasted with the color due to the basic fuchsin in the wedge.

4. That a magnifier and an illuminator be added to the scale in order to make it easily readable in the dark.

5. That a ratchet be placed on the handle of the wedge so that a recording may be fixed.

Dr. R. Denton Smith aided me in the study of the group of syphilitic patients.

Clinical Notes

TRACHOMA IN WRESTLERS

WILLIAM ZENTMAYER, M.D., PHILADELPHIA

In the fall of 1930 there was referred to me by Dr. Francis H. Lee of Richmond, Va., a professional wrestler with a badly inflamed right eye. The patient was on a circuit and had first noticed the trouble with his eye while he was in St. Louis about twelve days before his visit to me. He was treated there by an oculist who made a diagnosis of trachoma and prescribed a solution of a copper salt which, from the reaction following its use, the patient thought too strong. From St. Louis he went to Richmond and there saw Dr. Lee who also considered the disease trachoma.

When I saw him his eye had all the clinical appearances of a recent severe trachomatous infection. Papillary hypertrophy was marked. The bulbar conjunctiva was unusually congested, owing probably to the instillation of the solution of the copper salt an hour before. The limbal tissue was thickened and vascular. There was moderate ptosis. The patient made but the one visit, and a bacterial study was not obtained.

The patient was a college graduate, 31 years of age; he had been an athletic coach at a university in New York, and had only recently taken up professional wrestling. He was aware of the prevalence of trachoma among wrestlers and was not surprised when this diagnosis was made. His home was in New England, and he was given the name of an oculist in his home town and advised to consult him, but a communication from this physician a short time later showed that he had not done so.

A letter from Dr. Lee stated that the referee of wrestling matches in Richmond had told him that he had contracted trachoma while wrestling and had been under treatment for a long period. Dr. Lee also stated that a dentist in Richmond, a wrestling enthusiast, had informed him that wrestlers frequently have trachoma.

In 1922 the late Dr. James M. Patton, with the late Dr. Harold Gifford, saw eight cases of trachoma occurring among professional wrestlers of national reputation and others of less repute. The patients' statements made it seem probable that all the cases were indirectly traceable to the same person. The explanation of the mode of transmission was that in the course of wrestling matches the contestants perspired freely, and that when the referee was not looking too closely

Read before the Section on Ophthalmology of the College of Physicians, Philadelphia, Nov. 20, 1930.

This report is published at this time because of the interest of the National Society for the Prevention of Blindness in this subject.

they often massaged the opponent's eyes. This, Patton suggested, was done to produce unconsciously the oculocardiac reflex.

Patton stated that he was told of several other members of that profession similarly affected, but that he received no replies to his letters of inquiry to them concerning the condition of their eyes.

Dr. S. L. Baron, who makes the physical examination of wrestlers before a bout in Philadelphia, assures me that it includes examination of the eyes, and that in some cases he has seen a suspicious condition of the conjunctiva.

It is now twelve years since Patton[1] recorded his cases in the *American Journal of Ophthalmology*. To my knowledge no similar references have since appeared in the literature, and as inquiry reveals that Patton's observation is now little known, it seems proper to make this report so as to call the attention of ophthalmologists again to this mode of transmission of trachoma in order that they may bring it to the notice of the health authorities of their respective communities in an endeavor to eliminate this source of infection.

Since I reported the foregoing instance I saw a second case. On Oct. 25, 1933, there was referred to me by Dr. Roy Deck of Lancaster, Pa., a college student, aged 20 years. The patient stated that six weeks previously, a few days after wrestling with a professional, his left eye felt irritated, and that the following day the lids were swollen and there was a slight secretion from the conjunctiva. One week later the right eye was similarly affected. There was marked bulbar congestion. The entire tarsal and reflected conjunctiva was thickened and velvety. There was both follicular and papillary hypertrophy. The upper limbus was vascular. The biomicroscope showed that the limbal tissue was greatly thickened, and there were a number of superficial gray points in the corneal epithelium.

1. Patton, J. M.: Am. J. Ophth. **5**:545 (July) 1922.

THE "MYODISC" LENS AND BIFOCAL LENS

THEODORE E. OBRIG, A.B., NEW YORK

Most ophthalmologists have been confronted with the complaints of extremely myopic patients who object to the wearing of heavy ugly lenses. There has always been a need for a light and finer appearing lens to meet the needs of these patients.

During the past year I have used over one hundred pairs of a new type of lens for high myopia in corrections over 8 diopters. The lens has been named the "myodisc," suggesting a myopic disk lens. It was originally presented as a cosmetic lens. However, it has proved to have many other desirable qualities.

The "myodisc" is made in the form of a flat plano lens with a small concave disk ground in the center. The disks vary in size from 20 to 25 mm. The higher power lenses have the smallest disks. I have been

Fig. 1.—"Myodisc" single vision lenses (—20 diopters). Note the lack of distortion of the orbit and temple.

Fig. 2.—Comparative thickness of the "myodisc" and the old style lens of the same strength.

Fig. 3.—Both types of lenses (—20 diopters) showing the difference in thickness.

using and recommend a disk of 25 mm. for powers from — 8 to — 15 diopters, one of 22 mm. for powers from — 15.25 to — 18 diopters and one of 20 mm. for any corrections over — 18 diopters. These sizes have proved to give the patient a large enough field for general wear.

The lens had to be ground free from aberration, for the disk was so small that all of it had to be usable. The problem was that of polishing the plano band about the disk in such a manner as to do away with any turned edge at the junction of the two. The turned edge was the principal cause of the aberration.

The method of manufacture is new and has never been used before. It enables one to produce a lens eliminating the usual marginal and surface aberration of a concave disk when surrounded by a plano band. The cylinder is ground on the outside surface.

The cosmetic effect of the lens is marked. The lens weighs only one-third that of a double concave lens. It will be noticed in the illustration showing the relative thickness of two 20 diopter lenses that the

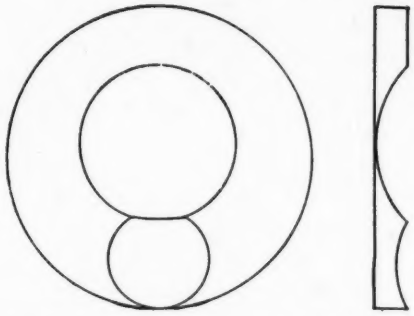


Fig. 4.—Diagram of the "myodisc" bifocal lens.

new "myodisc" is barely thicker than the ordinary white metal rim holding it. These results are obtained in regular crown glass.

Scientifically the "myodisc bifocal" is more important than the single vision lens, for it is the only optically perfect bifocal lens for high myopia with no heavy base down prism on the reading segment.

On the "myodisc bifocal" a second smaller concave disk of the same type is ground, having the same properties and located below the first larger disk. This second disk has an independent optical center affected in no way by the distance disk and is used as a reading segment. The upper part of the reading disk is ground into the upper disk in such a way as to produce a knife edge division with no aberration.

Because of the separate independent reading disk, a bifocal lens for high myopia with an actual optical center on the reading segment and a reading segment not affected by the distance sphere, free from chromatic aberration and usable over its entire area, is produced.

The Bausch & Lomb Optical Company of Rochester, N. Y., have consented to produce these lenses and will make them available to any one desiring to use them.

REFRACTIVE CHANGES IN ACUTE CHALAZIA

F. H. NEWTON, M.D., DALLAS, TEXAS

Rather sudden and extensive refractive changes in the presence of acute chalazia of the upper lids have been observed in three cases during the last two years. Careful examination and extended observation did not reveal any cause other than the influence of the chalazia.

REPORT OF CASES

CASE 1.—A white man, aged 43, was first seen on Sept. 17, 1928, at which time the vision was normal in each eye with +0.25 D. sph., +0.37 D. cyl., axis 25 for the right eye and +0.25 D. sph., +0.25 D. cyl., axis 90 for the left. A cycloplegic was not used. On Dec. 22, 1931, the vision in each eye was normal with +0.25 D. cyl., axis 180 in the right and +0.25 D. cyl., axis 145 in the left eye with an addition of +0.75 sph. for reading. On Aug. 8, 1932, the patient came in with a subacute chalazion of moderate size near the center of the left upper lid. This chalazion was removed in the usual way. The patient complained of some blurring in the left eye but had vision of 20/20 with +0.25 D. cyl., axis 180. He left town but returned on Sept. 9, 1932, stating that in the interim he had had two acute chalazia on the left upper lid opened. On his visit this lid was still somewhat swollen and moderately red but there were no localized areas requiring opening or removal. The vision in the left eye without glasses was 20/70. No definite changes in the media or fundus were noted, aside from a slight engorgement of the retinal veins; the field was moderately contracted for colors, and no scotomas were present. A complete physical examination gave normal results. The retinoscopic examination of the left eye required +2.50 D. sph. With homatropine cycloplegia the right eye showed 20/20 vision with +0.50 D. sph. and +0.25 D. cyl., axis 180, and the left, 20/30 vision with +3.00 D. sph., +0.25 D. cyl., axis 180. Thinking that possibly I was dealing with a large amount of latent hyperopia in the left eye and that the recurrent chalazia were due to insufficient correction, I prescribed +2.50 D. sph., +0.25 D. cyl., axis 180 for this eye. The patient was told of the unusual condition and requested to report for further observation. I never saw him again but a confrère reported that on Dec. 28, 1932, under homatropine cycloplegia the vision was 20/20 in each eye with +0.50 D. sph., +0.25 D. cyl., axis 180 in the right and +0.50 D. sph., +0.25 D. cyl., axis 180 in the left.

CASE 2.—A white man, aged 45, was first seen on Aug. 4, 1931, with a small hordeolum on the right upper lid. The vision was normal in each eye with +1.25 D. sph. for distance and +0.75 D. sph. added for reading. The correction was the same for each eye. On April 19, 1933, a refraction test showed normal vision in each eye with +1.00 D. sph., +0.25 D. cyl., axis 170 in the right and +0.75 D. sph., +0.25 D. cyl., axis 20 in the left with +1.75 D. sph. added for reading. On this date an acute chalazion on the right lower lid was opened. I saw the patient next on Oct. 3, 1933, with a large, subacute chalazion near the center of the right upper lid and extending to the margin of the lid. A test showed a vision of 20/25 with +2.50 D. sph., +0.50 D. cyl., axis 165 in the right eye; there were no changes in the left eye. On Oct. 4, 1933, under homatropine cycloplegia the right eye showed a vision of 20/25 with +3.00 D. sph. and +0.50 D. cyl., axis 180; in the left eye the vision was 20/20 with a +1.25 D. sph., +0.25 D. cyl., axis 30. In

the light of my experience with case 1 I explained the condition to the patient and advised waiting before giving a prescription. However, on Oct. 5, 1933, he insisted on a temporary lens for the right eye, and +2.50 D. sph., +0.50 D. cyl., axis 180 with +2.00 D. sph. added for reading was prescribed. On Oct. 20, 1933, he returned with all the swelling in the right upper lid gone; the vision was much blurred with his recently prescribed lens. The test showed a vision of 20/20 in the right eye with +1.25 D. sph., +0.37 D. cyl., axis 180. He experienced no further trouble after this change was made.

CASE 3.—A white woman, aged 30, applied for examination on Dec. 30, 1933. She had had recurrent multiple chalazia on both right eyelids. On examination four subacute chalazia were found near the margin of the right upper lid with considerable swelling of this lid. The vision in this eye was 20/60 with +1.50 D. sph., +0.37 D. cyl., axis 90 under homatropine cycloplegia. Under the same conditions, the vision in the left eye was normal with +0.75 D. sph., +0.25 D. cyl., axis 90. Otherwise the right eye was normal aside from a slight fulness of the retinal veins. A complete physical examination gave negative results. The chalazia were removed from the right upper lid on December 31. On Jan. 18, 1934, the swelling of this lid had subsided and the vision was $20/20^{-2}$ with +0.75 D. sph., +0.37 D. cyl., axis 90. The patient has had no further visual trouble.

The cause of these refractive changes is uncertain, but probably they are due to the pressure of the chalazia on the eyeball. In all three cases the chalazia were in a position to exert the maximum pressure on the ball, being located centrally and near the margin of the lid. If this explanation is correct, however, one would expect such changes to occur more frequently.

Personal communications from Dr. Harvey Howard and Dr. Sanford Gifford advance the theory of pressure as the cause of the condition. Dr. Gifford stated:

> One would have to imagine that the chalazion was in such a position as to press straight back on the center of the cornea when it was turned up during sleep and that this particular eye was lacking in elastic tissue, so as to maintain the flattened shape for some time. I suppose the reason more eyes do not show the same change is that their normal elasticity allows them to regain their shape rapidly on awakening.

Pressure would hardly account for the decided diminution in vision, especially in case 3. This explanation does not seem to me entirely satisfactory, but I have no better one to offer.

Ophthalmologic Review

EDITED BY DR. FRANCIS HEED ADLER

LOCAL ANESTHESIA

C. S. O'BRIEN, M.D.
IOWA CITY

Local anesthesia is preferable to general narcosis in practically all ophthalmic operations. Successful administration comes only through study of the sensory nerve supply to the globe and adnexa and through repeated use of such anesthesia.

INDICATIONS

Local anesthesia may be employed, with few exceptions, in any operation on a juvenile or an adult patient. It is frequently advisable to administer a sedative an hour or two prior to operation, especially to apprehensive and nervous persons. General anesthesia is more desirable for young children and for older children in whom the element of fear is uncontrollable.

ADVANTAGES

The following advantages of local over general anesthesia may be enumerated:

Safety.—The greater safety of properly chosen local anesthetics is well established. For injection into the tissues it is advisable to use only comparatively small amounts of drugs of low toxicity. Such anesthesia is much safer than general anesthesia for patients with cardiac disturbances, nephritis, arteriosclerosis and other general diseases. Rarely an idiosyncrasy to some one anesthetic may exist, but usually the patient is aware of such a condition and another drug may be substituted.

Cooperation of the Patient.—Cooperation is especially desirable in ophthalmic operations and may be secured only with local anesthesia.

Sterility of the Operative Field.—There is no danger of contamination from the hands of an anesthetist or from the appliances necessary for general anesthesia. Furthermore, the danger of contamination from vomitus is lessened.

From the Department of Ophthalmology, College of Medicine, State University of Iowa.

Unobstructed Operative Field.—Masks, inhalors, tubes and other instruments employed in general anesthesia often obstruct the field and make an operation more difficult.

Lessened Bleeding.—Small amounts of epinephrine hydrochloride combined with the anesthetic solution contract the blood vessels and lessen bleeding. In nitrogen monoxide or ethylene, and sometimes in ether narcosis, the vessels are engorged.

Infrequency of Vomiting.—Vomiting is not so often encountered, either during or after an operation, as with general anesthesia. Consequently contamination of the wound is less frequent, and undue strain on sutures is rare.

Speed.—A series of operations is more rapidly consummated, unless two general anesthetists are available.

Postoperative Quietude.—Patients are conscious and less excited, and therefore less liable to damage of the wound.

Absence of Postoperative Complications.—Pneumonia, nephritis and other conditions which may follow general narcosis do not occur as a result of the administration of local anesthetics.

DISADVANTAGES

Uncontrollable fear, occurring as a rule only in children, makes it impossible to work under local anesthesia.

In highly inflamed tissues it is more difficult to secure complete loss of sensation, but, as a rule, this can be done by proper administration of the anesthetic.

ANESTHETICS

Numerous anesthetics are available. It is advisable to select only three or four drugs and to become familiar with their actions and possibilities through constant usage. The following characteristics are desirable:

1. The production of efficient and lasting anesthesia.
2. Low toxicity in the necessary dosage.
3. The production of vasoconstriction, or at least only slight vasodilatation.
4. Stability on sterilization.
5. Failure to produce mydriasis. In some operations a dilated pupil is desirable, e. g., in the intracapsular extraction of cataract.
6. The production of little or no disturbance of the corneal epithelium.

There is no drug which meets all of these requirements, but cocaine hydrochloride, butyn and procaine hydrochloride are efficient and trustworthy.

All solutions must be fresh and sterile and should be made up and sterilized in the autoclave or by boiling immediately before use. It is preferable to employ for injections those which are marketed in small ampules, since they cannot deteriorate from contact with the air, are sterile and are always ready for use.

A small amount of epinephrine hydrochloride administered with the anesthetic augments and prolongs anesthesia, prevents too rapid absorption into the general circulation and provides a comparatively dry field of operation. A solution of suprarenin bitartrate may be instilled in operations in which mydriasis and extreme vascular constriction are desirable, e. g., in the intracapsular extraction of cataract. This drug is obtainable in ampules containing 0.91 Gm. The contents of the ampule are dissolved in 4 cc. of distilled water, and the solution is ready for use. It deteriorates rather rapidly and is stable for approximately only one week.

For injection, accurately calibrated, all glass, light-weight syringes are preferable. They are sterilized by dry heat, steam or boiling. Needles should be of fine gage (24 or 26), sharp and of proper length.

SEDATIVES

In preparation for an operation under local anesthesia a sedative is desirable; it tends to dull sensation, allay fear and quiet the patient. A mixture of the hydrochlorides of the opium alkaloids, ⅓ grain (0.02 Gm.), by hypodermic injection or sodium bromide, 30 grains (0.9 Gm.), in milk by mouth is perhaps as good as any available preparation. It is unwise to prescribe morphine since its administration is often followed by vomiting.

METHODS OF ADMINISTRATION

Several methods are employed in the administration of local anesthetics. Only one method may be needed in preparation for an operation, but usually a combination of two or more is necessary for complete anesthesia.

Instillation.—A weak anesthetic solution is instilled into the conjunctival sac.

Anesthesia: After repeated instillations there is complete loss of sensation in the conjunctiva and cornea, with partial loss in the iris and ciliary body.

Indications: Instillation may be used in operations on the globe or conjunctiva. Instillations alone produce sufficient anesthesia for minor procedures on the conjunctiva and cornea, but if the globe is to be opened, subconjunctival or subconjunctival and retrobulbar injections are also employed.

Drugs: Cocaine hydrochloride 5 per cent in sterile distilled water is an excellent anesthetic; it has a moderate vasoconstrictor action but dilates the pupil and softens the corneal epithelium.

Butyn 2 per cent in sterile physiologic solution of sodium chloride does not affect the pupil, but the anesthesia is not as deep as with cocaine and there is some vasodilatation.

Administration: One or 2 drops of the solution is placed at the upper limbus and allowed to flow over the cornea, and the lids are closed immediately. For complete anesthesia five instillations at two minute intervals are advisable. At alternate minutes 1 : 1,000 epinephrine hydrochloride is instilled. The lids should remain closed, since exposure augments the deleterious action of the anesthetic on the corneal epithelium.

Topical Application.—A strong anesthetic solution or the drug in its pure state is applied over a small localized area.

Anesthesia: Moderately deep loss of sensation is produced at the point of application.

Indications: This method may be used in minor operations on a small localized area of the cornea, conjunctiva or other tissue.

Drug: Cocaine hydrochloride crystals are the most satisfactory preparation.

Administration: A small applicator is wound with cotton, dipped into 1 : 1,000 epinephrine hydrochloride and then into cocaine hydrochloride crystals and held for a few seconds against the tissues at the site of operation. Anesthesia is complete in five minutes.

Subconjunctival Injection.—A weak anesthetic solution is injected under the conjunctiva of the globe or the fornix.

Anesthesia: Injection under the bulbar conjunctiva completely anesthetizes the conjunctiva and sclera in the area of injection, the iris and the ciliary body and produces almost complete loss of sensation in the nerves of the suprachoroidea. Injections into the fornix produce deep anesthesia of the conjunctiva and of underlying structures in the lid and fornix.

Indications: Injection under the bulbar conjunctiva is indicated in all operations on the globe. Subconjunctival injections in the area of the fornix are employed in expression of trachoma follicles, resection of the accessory portion of the lacrimal gland, advancement of the levator palpebrae superioris muscle and other operations in these areas.

Drugs: Procaine hydrochloride 2 per cent, in physiologic solution of sodium chloride, to each cubic centimeter of which is added 1 drop of 1 : 1,000 epinephrine hydrochloride, may be used. The 3 cc. ampules containing 0.06 Gm. of procaine hydrochloride and 0.00015 Gm. of an epinephrine compound are reliable and convenient.

Cocaine hydrochloride 1 per cent in sterile distilled water, to each cubic centimeter of which is added from 3 to 5 drops of 1:1,000 epinephrine hydrochloride, is employed in cataract extraction since it produces excellent anesthesia and the mydriasis necessary for the intracapsular operation. A 1 cc. ampule of cocaine hydrochloride 1 per cent with 5 minims of epinephrine hydrochloride is obtainable.

Administration: Following two instillations of 5 per cent cocaine hydrochloride or 2 per cent butyn, with a tuberculin syringe and a regular hypodermic needle from 0.2 to 0.3 cc. of the procaine-epinephrine or the cocaine-epinephrine solution is injected under the conjunctiva, anterior to the tendinous attachment of one or more of the rectus muscles. Gentle massage over the closed lids soon distributes the solution; it infiltrates into the tissues, and deep anesthesia occurs in from ten to twelve minutes. Procaine-epinephrine solution is injected under the fornix for operations in that area.

Intramuscular Injection.—A weak anesthetic solution is injected into the belly of an extra-ocular muscle.

Anesthesia: Deep anesthesia of the muscle is produced. Along with the loss of sensation there is more or less akinesis since some sensory nerve fibers run forward in the third, fourth and sixth cranial nerve trunks and their branches.

Indications: Intramuscular injection may be used in operations on the extra-ocular muscles.

Drug: Procaine hydrochloride 2 per cent in physiologic solution of sodium chloride, to each cubic centimeter of which is added 1 drop of 1:1,000 epinephrine hydrochloride, is the most satisfactory preparation. The previously mentioned 3 cc. ampules of procaine hydrochloride and an epinephrine compound are excellent.

Administration: Following five instillations of cocaine hydrochloride 5 per cent or butyn 2 per cent, the intramuscular injection is made with a 2 cc. syringe and a 26 gage needle of 25 mm. (1 inch) length. With a moderately heavy mouse tooth forceps one grasps the conjunctiva and the tendon of the muscle near its insertion into the sclera and pushes the needle through the conjunctiva along the tendon and backward into the belly of the muscle; 2 cc. of the procaine-epinephrine solution is injected as the needle is slowly withdrawn. The injection must be made into the muscle; otherwise the result is unsatisfactory. Anesthesia is complete in from ten to twelve minutes.

Tissue Infiltration.—Subcutaneous and deep injections of a weak anesthetic solution are made into the tissues in the area of operation.

Anesthesia: There is loss of sensation in all the structures infiltrated by the solution.

Indications: This method may be used in operations on the orbit, the lids, the lacrimal sac and adjoining areas, but on account of dis-

tortion of the tissues and disturbance in the anatomic relations, it is usually not as satisfactory as nerve block at a site remote from the field of operation.

Drug: Procaine hydrochloride 1 per cent, to each 2 cc. of which is added 1 drop of 1:1,000 epinephrine hydrochloride, is the most satisfactory preparation.

Administration: With a 5 cc. syringe and a 26 gage needle of 25 mm. (1 inch) length, injections of the procaine-epinephrine solution are made into the subcutaneous and deeper structures at the site of operation. Not over 10 cc. of the solution is used. Anesthesia is complete in from eight to ten minutes.

Retrobulbar Injection.—A weak anesthetic solution is injected directly behind the eyeball.

Anesthesia: The ciliary nerves are blocked immediately behind the globe, and there is deep anesthesia of the eyeball and orbital structures within the muscle cone. If the solution is improperly administered and discharged into a muscle rather than within the muscle cone, temporary paresis of one or more of the rectus muscles follows.

Indications: This method may be used in operations in which deep anesthesia of the eyeball is desired.

Drug: Procaine hydrochloride 2 per cent, to each cubic centimeter of which is added 2 drops of 1:1,000 epinephrine hydrochloride, is the most satisfactory preparation.

Administration: The retrobulbar injection of procaine-epinephrine solution is made following five instillations of 5 per cent cocaine hydrochloride or 2 per cent butyn. A 2 cc. syringe and a 26 gage needle of 37.5 mm. (1½ inch) length are used. With the patient looking upward and nasally and the lower lid pulled downward, the needle is inserted in the inferolateral fornix between the inferior and lateral rectus muscles and pushed through the tissues toward the entrance of the optic nerve into the globe; when the point reaches this region 1 cc. of the solution is injected. It is almost impossible to injure the optic nerve by such a procedure, but in extremely rare instances retrobulbar hemorrhage occurs. Anesthesia is complete in from eight to ten minutes.

Nerve or Ganglion Block.—This method consists in the infiltration with a weak anesthetic solution of a nerve trunk or ganglion at a point remote from the site of operation; the injection must be made into or very near the nerve or ganglion.

Such injections produce excellent anesthesia and do not waterlog the tissues or disturb the anatomic relations in the operative field. There are several branches of the trigeminus nerve which supply sensation to the globe and adnexa, and in most operations it is necessary to block two or more of these branches, since their terminal filaments overlap and one area may be supplied by two or more nerves. A 5 cc. syringe

with a 26 gage needle of 25 mm. (1 inch) length is used for all injections except ciliary ganglion block, for which a 37.5 mm. (1½ inch) needle is necessary. Procaine hydrochloride 2 per cent, with 1 drop of 1 : 1,000 epinephrine hydrochloride per cubic centimeter, is employed. Not over 5 or 6 cc. of the solution is used. Anesthesia is complete almost immediately if the injection is made close to the nerve.

Supra-Orbital Nerve Block. Anatomy: The supra-orbital nerve, a branch of the ophthalmic, emerges from the upper nasal portion of the orbit through the supra-orbital notch or foramen. This notch or

Fig. 1 (adapted from Spalteholz).—Orbital margin and septum, with sensory nerves: (*a*) supra-orbital, (*b*) frontal, (*c*) supratrochlear, (*d*) infratrochlear, (*e*) infra-orbital, (*f*) zygomaticofacial and (*g*) lacrimal. These nerves may be blocked as they emerge from the orbital margin or bone.

foramen is usually present and is situated from 20 to 25 mm. from the midline of the glabella at the junction of the inner and middle thirds of the superior orbital margin; it lies at the point where the sharp outer two thirds meet the rounded inner third of the orbital rim. The nerve is superficial, lying just under the skin and orbicularis muscle.

Anesthesia: The central portion of the upper lid, the forehead and the anterior part of the scalp are anesthetized.

Administration: The supra-orbital notch or foramen is located with the index finger, and into or just over it 1 cc. of the procaine-epineph-

rine solution is injected. The injection is made at the anterior surface of the orbital margin, in front of the orbital septum.

Frontal Nerve Block. Anatomy: The frontal nerve is a branch of the supra-orbital which sometimes leaves the latter nerve after it emerges from the orbit but occasionally branches off farther back and runs forward to emerge from the orbit a few millimeters nasal to the supra-orbital.

Anesthesia: The upper lid, forehead and anterior portion of the scalp are anesthetized.

Administration: Following injection of the procaine-epinephrine solution into the region of the supra-orbital nerve at the supra-orbital notch, the needle is partially withdrawn and carried 5 or 6 mm. nasally along the bony orbital rim, and another injection of from 0.5 to 1 cc. is made.

Supratrochlear and Infratrochlear Nerve Block. Anatomy: The supratrochlear nerve, a branch of the frontal, and the infratrochlear, a branch of the nasociliary, run immediately above and below the trochlea of the superior oblique muscle. Frequently an anastomosis is formed just anterior to this point. The trochlea is situated in the superior nasal angle of the orbit, approximately 1 cm. behind the orbital margin.

Anesthesia: Supratrochlear block anesthetizes the medial part of the upper lid, the lower medial portion of the forehead and the root of the nose. Infratrochlear block anesthetizes the medial portions of both lids, the caruncle, the lacrimal sac and the ala of the nose.

Administration: The trochlea is recognized by palpation as a small hard elevation with a moderately sharp temporal edge. Both nerves are infiltrated by a single injection of 1 cc. of the procaine-epinephrine solution just anterior to the trochlea.

Lacrimal Nerve Block. Anatomy: The lacrimal nerve, a branch of the ophthalmic, emerges from the orbit immediately above the lateral canthal ligament, in the small area between the lateral orbital tubercle of the zygomatic bone below and the zygomaticofrontal suture above.

Anesthesia: The lateral portion of the upper lid and the region of the lateral canthus are anesthetized.

Administration: The lateral canthal ligament and the underlying lateral orbital tubercle are located by palpation; the needle is passed to the bony rim of the orbit just above these structures, and 1 cc. of the procaine-epinephrine solution is injected.

Zygomaticofrontal Nerve Block. Anatomy: This small nerve is a filament from the zygomatic branch of the maxillary nerve. It emerges through a minute foramen in the zygomatic bone approximately 1 cm. lateral to the inferior lateral angle of the orbit.

Anesthesia: The lateral portion of the lower lid and the cheek are anesthetized.

Administration: One cubic centimeter of the procaine-epinephrine solution is injected over the bone at a point 1 cm. lateral to the inferior lateral angle of the orbit.

Infra-Orbital Nerve Block. Anatomy: This large nerve, a continuation of the maxillary, courses through the infra-orbital canal and emerges from the face through the infra-orbital foramen, an opening in the maxillary bone situated from 7 to 8 mm. below the center of the inferior orbital margin and from 25 to 30 mm. lateral to the midline of the face. The long axis of the canal most frequently extends in an upward, backward and temporal direction, but it is variable and may extend upward, backward and nasally, or upward and backward.

Anesthesia: The lower lid, the medial and lateral portions of the upper lid, the lacrimal sac, the side of the nose and the temporal region are anesthetized.

Administration: The bony prominence immediately below the center of the inferior orbital margin is located by palpation. A 2 cm. fine gage needle, pointed upward and temporally in the direction of the lateral canthus, is entered just lateral to the wing of the nose and carried down to the bone. A few minims of procaine-epinephrine solution is injected, a search is made for the foramen, then the needle is plunged into the infra-orbital canal, and 1 cc. of the solution is injected. It is not always possible to enter the foramen, but an injection at the opening or immediately above it blocks the nerve.

Ciliary Ganglion and Long Ciliary Nerve Block. Anatomy: The ciliary ganglion is situated between the optic nerve and the lateral rectus muscle, near the apex of the orbit; it lies approximately 15 mm. behind the globe and from 37 to 38 mm. back of the lateral orbital margin. The short ciliary nerves run forward from this ganglion to the eyeball, and the two long ciliary nerves, which are branches of the nasociliary, are in close proximity to the ganglion.

Anesthesia: There is loss of sensation in the cornea, sclera, choroid, iris and ciliary body.

Administration: Following from three to five instillations of cocaine hydrochloride 5 per cent or butyn 2 per cent, the needle is passed through the conjunctiva and other tissues at the temporal side of the inferior fornix; it is pointed toward the orbital apex, and an attempt is made to guide it between the lateral rectus muscle and the optic nerve. The injection of 2 cc. of procaine-epinephrine solution is made when the needle reaches a depth of from 38 to 40 mm. from the orbital margin. The solution diffuses through the tissues and into the ciliary ganglion and long ciliary nerves. Anesthesia is usually complete in from ten to twelve minutes.

AKINESIS

In cataract extraction and at times in other intra-ocular operations it is an advantage if the muscles of lid closure are partially or completely paralyzed. Such paralysis prevents squeezing, and vitreous is seldom lost.

Several muscles come into action in forcible closure of the lids: the orbicularis oculi, frontalis, corrugator supercilii, pyramidalis nasi and quadratus labii superioris. They are all supplied through the temporal, malar and infra-orbital branches of the temporofacial division of the facial nerve.

Several methods of injection are employed to induce paralysis of these muscles. A simple and satisfactory method is as follows: The zygomatic arch is located with the index finger, and its under side is followed backward to a point just in front of the tragus of the ear.

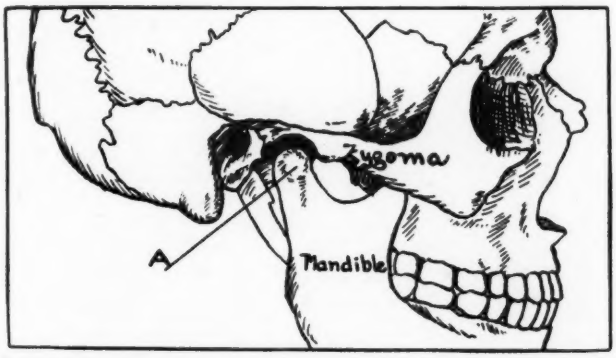


Fig. 2 (from Spalteholz).—The injection for akinesis of the temporofacial division of the facial nerve is made over the anterior half of the condyloid process of the mandible (*A*). Injections over the posterior half of the condyloid process fail to produce akinesis.

With the tip of the finger in this position the bony condyloid process of the mandible is palpable. The patient is asked to open the mouth, and the condyloid process is felt to slip forward under the finger. The mouth is closed, and the process slips backward. A short needle (⅝ inch [1.5 cm.]) is passed down to the condyloid process at a point anterior to its center, and from 1 to 2 cc. of 2 per cent procaine hydrochloride is injected; the last portion of the solution is injected as the needle is slowly withdrawn. The needle must strike the anterior and not the posterior portion of the condyloid process; otherwise akinesis will not result.

ANESTHESIA IN SPECIFIC OPERATIONS

The following outline indicates the methods that may be used for various operations:

1. For intra-ocular operations, e. g., cataract extraction, iridectomy, iridencleisis, iridotasis, corneoscleral trephination, sclerectomy, cyclodialysis, cauterizing operations for retinal detachment and removal of intra-ocular foreign bodies:

Instillations of 5 per cent cocaine hydrochloride or 2 per cent butyn (five instillations at two minute intervals). Cocaine is employed unless there is a contraindication to mydriasis.

Instillations of 1:1,000 epinephrine hydrochloride (three instillations, i. e., one following the first, second and third instillation of cocaine or butyn). If wide mydriasis is desired, as in intracapsular cataract extraction, suprarenin bitartrate is substituted.

Subconjunctival injection below the lower limbus of from 0.2 to 0.3 cc. of 2 per cent procaine hydrochloride containing 1 drop of epinephrine hydrochloride. If wide mydriasis is desired, one may substitute 1 per cent cocaine hydrochloride containing 1 or 2 drops of epinephrine hydrochloride. This injection is made following the second instillation of cocaine hydrochloride or butyn.

Retrobulbar injection of 1 cc. of 2 per cent procaine hydrochloride containing 2 drops of epinephrine hydrochloride. The injection is made following the fifth instillation of cocaine hydrochloride or butyn.

2. For repair of iris prolapse:

Instillations of 5 per cent cocaine hydrochloride and epinephrine hydrochloride.

Subconjunctival injection of 1 per cent cocaine hydrochloride with epinephrine hydrochloride near the site of prolapse.

Topical application of cocaine crystals to the conjunctiva over the site of prolapse, and even to the prolapsed iris in some cases.

3. For discission:

Instillations of 5 per cent cocaine hydrochloride and an epinephrine compound.

Subconjunctival injection of 1 per cent cocaine hydrochloride with epinephrine hydrochloride.

4. For peritomy and removal of tumors of the limbus:

Instillations of 5 per cent cocaine hydrochloride and epinephrine hydrochloride.

Subconjunctival injection of 1 per cent cocaine hydrochloride near the site of operation.

5. For paracentesis of the anterior chamber:

Instillations of 2 per cent butyn and epinephrine hydrochloride.

Subconjunctival injection of 2 per cent procaine hydrochloride with epinephrine.

6. For enucleation or evisceration:

Instillations of 5 per cent cocaine and epinephrine hydrochloride, as in intra-ocular operations.

Intramuscular injection of 0.5 cc. of 2 per cent procaine hydrochloride containing 1 drop of epinephrine hydrochloride per cubic centimeter into the belly of each rectus muscle. These injections follow the last instillation of cocaine.

Ciliary ganglion block with from 1 to 2 cc. of 2 per cent procaine hydrochloride containing 2 drops of epinephrine hydrochloride.

7. For operations for strabismus, e. g., advancement, resection, recession and tucking:

Instillations of 5 per cent cocaine hydrochloride or 2 per cent butyn and epinephrine hydrochloride, as in intra-ocular operations.

Intramuscular injection, following the last instillation of cocaine hydrochloride or butyn, of 2 cc. of 2 per cent procaine hydrochloride containing 2 drops of epinephrine hydrochloride.

8. For extirpation of the lacrimal sac:

Supratrochlear and infratrochlear nerve block with 2 per cent procaine hydrochloride containing 1 drop of epinephrine hydrochloride per cubic centimeter.

Infra-orbital nerve block.

Topical application of cocaine crystals to the mucosa of the nasolacrimal duct. This is done following removal of the sac, in preparation for curettage of the duct.

9. For resection of the accessory portion of the lacrimal gland:

Instillations of 5 per cent cocaine hydrochloride and epinephrine hydrochloride.

Lacrimal nerve block with 2 per cent procaine hydrochloride containing epinephrine hydrochloride.

Subconjunctival injection into the lateral portion of the upper fornix of 1 cc. of 2 per cent procaine hydrochloride containing epinephrine hydrochloride.

10. For operations for pterygium, e. g., transplantation:

Instillations of 5 per cent cocaine hydrochloride and epinephrine hydrochloride.

Subconjunctival injection of 0.2 cc. of 1 per cent cocaine hydrochloride containing 1 drop of epinephrine hydrochloride over the attachment of the inferior rectus tendon.

11. For chalazion:

For opening, evacuation and curettage from the conjunctival side, instillations of 5 per cent cocaine hydrochloride, followed by topical applications of cocaine crystals to the conjunctiva over the chalazion and further topical applications to the inside of the tumor after evacuation.

For complete removal by dissection from the skin side, blocking of the nerves supplying the area with 2 per cent procaine hydrochloride containing epinephrine hydrochloride (e. g., if the chalazion is situated

at the lateral side of the upper lid the supra-orbital and lacrimal nerves are blocked). Tissue infiltration around the affected area with 2 per cent procaine hydrochloride is also satisfactory, but it disturbs the tissues and dissection is more difficult.

12. For symblepharon:

Instillations of 5 per cent cocaine hydrochloride and epinephrine hydrochloride.

Subconjunctival injection, in the affected area, of 2 per cent procaine hydrochloride containing epinephrine hydrochloride.

13. For canthotomy:

Instillations of 5 per cent cocaine hydrochloride and epinephrine hydrochloride.

Injection into the tissues at the lateral canthus of 2 per cent procaine hydrochloride containing epinephrine hydrochloride.

14. For expression of trachoma follicles, e. g., lid rolling and other operations:

Instillations of 5 per cent cocaine hydrochloride and epinephrine hydrochloride.

Subconjunctival injection into the fornix of from 1 to 2 cc. of 2 per cent procaine hydrochloride containing epinephrine hydrochloride.

15. For ptosis:

(The anesthesia depends somewhat on the type of operation.)

Instillations of 5 per cent cocaine hydrochloride and epinephrine hydrochloride.

Supra-orbital and frontal nerve block with 2 per cent procaine hydrochloride containing epinephrine hydrochloride.

Supratrochlear nerve block

Lacrimal nerve block.

16. For plastic operations on the upper lid, e. g., entropion and ectropion:

Instillations of 5 per cent cocaine hydrochloride and epinephrine hydrochloride.

Supra-orbital and frontal nerve block with 2 per cent procaine hydrochloride containing epinephrine hydrochloride.

Supratrochlear nerve block.

Lacrimal nerve block.

Infra-orbital nerve block.

17. For plastic operations on the lower lid:

Instillations of 5 per cent cocaine hydrochloride and epinephrine hydrochloride.

Infra-orbital nerve block.

Infratrochlear nerve block.

Lacrimal nerve block.

18. Exenteration of the orbit, reconstruction of the socket and removal of orbital tumors:

Instillations of 5 per cent cocaine hydrochloride and epinephrine hydrochloride.

Blocking of each of the branches of the ophthalmic nerve with 2 per cent procaine hydrochloride containing epinephrine hydrochloride.

Ciliary ganglion block with 2 per cent procaine hydrochloride containing epinephrine hydrochloride.

Notes and News

UNIVERSITY NEWS

During the week of Jan. 14 to 19, 1935, at the Northwestern University Medical School, a course will be given on the diagnosis and surgical and nonsurgical treatment of strabismus, including dissection of the cadaver. Half the day will be spent in lectures and demonstrations and the other half day in the hospital, the orthoptic clinic and the laboratory of surgical anatomy. Attendance will be limited to fifteen persons, and accommodations for those desiring them will be arranged at the Lake Shore Athletic Club. Other courses in ophthalmology for larger or smaller groups will be given during the month of January by various teaching institutions in Chicago. For detailed information apply to Dr. Sanford Gifford, Northwestern University Medical School.

Abstracts from Current Literature

EDITED BY DR. WILLIAM ZENTMAYER

Anatomy and Embryology

PLASTOSOMES OF THE GROWING LENS. W. RAUH, Arch. f. Ophth. **130:** 213 (July) 1933.

Plastosome is another name for the granular elements of protoplasm and, therefore, a synonym for mitochondria, chondriosome and Altmann's granula. The author studied the plastosomes in the lenses of rats of various ages. The capsule of the lens, with the adhering portions of the cortex, and the rest of the cortex were studied separately. Osmic acid mixtures were used as fixatives; iron-alum-hematoxylin was used for staining the sections, which were 5 microns thick. With this method plastosomes were found in the lenses of rats up to the age of 3 years (the age limit of rats). The plastosomes were always present in the epithelium and in the cortex. In the nucleus the plastosomes disappeared shortly after birth. They had the shape of short comma-like rods. The areas in the tissue of the lens which contained plastosomes also synthesized indo-phenol. These facts are in accord with the presence of cell respiration of the lens during the entire life of the animal.

P. C. KRONFELD.

VARIATION IN THE HORIZONTAL AND VERTICAL DIAMETERS OF THE CORNEA AND IN THE SURFACE AREAS OF THE CORNEA AND SCLERA IN MEGALOCORNEA, NORMAL CORNEA AND MICROCORNEA. R. FRIEDE, Ztschr. f. Augenh. **81:** 132 (Aug.) 1933.

THE SURFACE AREAS OF THE CORNEA AND SCLERA IN THE EMBRYO AND THE NEW-BORN AND THEIR RELATION TO MEGALOCORNEA IN THE ADULT. R. FRIEDE, Ztschr. f. Augenh. **81:** 213 (Sept.) 1933.

The theme of the author in both these papers is that megalocornea is not a form of giant growth associated with enlargement of the eyeball, but that it is an increase in the surface area of the cornea at the expense of the sclera. Normal relations in the surface areas of these two structures may be expressed by the term "normal cornea, normal sclera." Megalocornea may be designated "macrocornea, microsclera" and microcornea (without microphthalmia) "microcornea, macrosclera."

In the first paper the author presents tables and graphs based on measurements with the Wessely keratometer of the corneal diameters in 10,940 eyes. The length of the horizontal diameter varied between 10 and 13.5 mm., with an average of 11.56 mm. The vertical diameter varied from 9 to 13 mm., with an average of 11.25 mm. The ratio of the corneal surface to that of the sclera is 1:4.76. If this ratio is approximated in any eye, one may speak of normal cornea, normal sclera.

In his second paper the author exhibits tables showing measurements of the cornea in the human embryo, new-born infant and child up to 5 years of age. Comparisons are made with the horizontal diameter of the eyeball. The ratio of the corneal to the scleral surface area is placed at 1 : 3.624, to be compared with the adult ratio of 1 : 4.76. Megalocornea, therefore, is the natural form in the embryo and the young child. A graph based on measurements of corneal and eyeball diameters in an ascending series of animals, human embryos, infants, young children and adults indicates that megalocornea is the natural form in animals and persists in a lessening degree in the human embryo and child.

H. L. BEGLE.

Aqueous Humor

THE AQUEOUS AND THE LENS METABOLISM: II. THE REDUCTION POTENTIAL OF THE AQUEOUS. H. MÜLLER, Arch. f. Augenh. **108:** 51, 1933.

Using an electrometric method Müller was able to obtain definite reproducible values of the aqueous reduction potential. When the aqueous has a p_H of 9.2 the average reduction potential is 55 millivolts. With a p_H of from 4.1 to 9.4 the reduction potential shows straight-line dependence on the hydrogen ion concentration.

Electrometric titration of the aqueous with iodine demonstrates the typical behavior of a reversible reduction system in a biologic medium, from which it may be concluded that the aqueous contains a reduction system.

The reduction potential can be changed by diluting the aqueous, which is interpreted by Müller as indicating that the reduction system of the aqueous is not a reversible one.

F. H. ADLER.

Bacteriology and Serology

A MICRO-ORGANISM FOUND FOR THE FIRST TIME ON THE HUMAN CONJUNCTIVA. A. GABRIÉLIDÈS, Ann. d'ocul. **170:** 683 (Aug.) 1933.

The number of saprophytic and pathogenic organisms found on the human conjunctiva is large, and their rôle in the causation of a large number of ocular infections is definitely established. They may provoke postoperative infections, especially following extraction of cataracts. It is for this reason that special preoperative care is taken, such as washing the conjunctiva, lacrimal sac and other parts.

The method used in isolating a gram-positive micro-organism which was at first associated with staphylococci and Bacillus xerosis and the cultural features of the micro-organism are described in detail. From a study of the different textbooks of ophthalmic bacteriology, Gabriélidès believes that this micro-organism has not been isolated before from the human conjunctiva, but he has not been able as yet to determine whether it is a new micro-organism.

S. H. MCKEE.

Conjunctiva

PERITOMY TO PREVENT GONORRHEAL COMPLICATIONS OF THE CORNEA. REPORT OF A CASE. K. HARTMANN, Klin. Monatsbl. f. Augenh. **91:** 249 (Aug.) 1933.

A man, aged 21, acquired a fulminant gonorrheal conjunctivitis in one eye a few days after the urethral infection became evident. The usual local therapy and milk injections were of no avail, and peritomy was followed by a deep corneal ulcer which terminated in destruction of the cornea, prolapse of the iris and a staphyloma involving about one fifth of the cornea. The staphyloma was removed and a plastic operation after Kuhnt was performed, during which the conjunctival flap was reenforced by episcleral tissue and the upper layers of the sclera. Full vision was obtained. Peritomy will not succeed in preventing a corneal ulcer, or in arresting an existing one; it should be nevertheless resorted to after milk injections have failed.

K. L. STOLL.

Comparative Ophthalmology

THE FORMATION OF ROSETTES IN THE RAT RETINA. K. TANSLEY, Brit. J. Ophth. **17:** 321 (June) 1933.

Tansley discusses abnormal conditions in which retinal rosettes are encountered, such as neuro-epitheliomas (blastomas) in the folded retinas of microphthalmic eyes and occasionally at the edges of colobomas, and the rosettes found in the retinas of human fetuses which have been treated with large doses of roentgen rays during the second month of their development. Rosettes have never been found in the retina of normally developed human eyes.

For the study of these conditions he used cultures of the rat retina. There is no doubt that the rosettes which appear in the retina both in tissue cultures and as a result of trephining the growing eye are formed from the neuro-epithelial layers as described by Flexner and Wintersteiner. In fact, it seems probable that in all cases the rosettes are produced from this part of the retina. Their formation appears to be essentially a feature of the developing retina, and probably never occurs in one that is fully differentiated. There is no stage of development that is especially favorable to their production. When rosettes are formed in a retina that is situated within the eye, abnormal growth of the neuro-epithelial layers might cause *inward* folding of the retina, for since the expanded tissue must be accommodated, it is probably easier for it to penetrate the inner layers of the retina than the hard fibrous layers of the choroid and sclera.

A serious objection to the view that rosettes are produced by an overgrowth of the neuro-epithelium is that mitoses have not been observed in the cells immediately surrounding the rosettes in any case in tissue cultures, although they are frequent in this part of the retina in the normally developing eye.

Under the conditions of the present work there certainly seems to be a definite relation between the maintenance of a normal intra-ocular pressure and the appearance of retinal rosettes.

The article is illustrated and contains a bibliography.

W. ZENTMAYER.

Congenital Anomalies

CONGENITAL PSEUDOPAPILLEDEMA. F. SPINELLI, Arch. di ottal. **40:** 237 (May) 1933.

A man of 22 years showed hyperopia of 22 diopters and a condition of the fundus resembling papilledema. This was due apparently to dense glial tissue on both disks, and was associated with white punctate deposits in the periphery resembling those of retinitis punctata albescens. The globes were very small, and vision with correction was 1/50 and 3/50. The fields showed some constriction, but hemeralopia was not present.

S. R. GIFFORD.

CONGENITAL ECTOPIA OF THE LENS INHERITED THROUGH FOUR GENERATIONS. H. ROESE, Ztschr. f. Augenh. **81:** 149 (Aug.) 1933.

The author states that congenital ectopia of the lens is of infrequent occurrence. Grob reported 18 cases in the Zurich eye clinic among 68,500 patients over a period of thirty-eight years. Ringelhan and Elschnig mentioned 23 cases among 192,000 patients attending the eye clinic at Prague in the last twenty-three years. In almost half of the reported cases there is evidence of inheritance, and the rarity of the deformity is emphasized if the affected families only are counted. The author outlines a genealogical tree showing 9 members with the deformity in four generations. Since most of the afflicted persons were observed over a long period of time, numerous complications appeared, and the author comments on changes in the position of the lens, luxation of the lens into the vitreous or anterior chamber, attacks of increased tension, scleral staphyloma and axial myopia with choroidal degeneration.

H. L. BEGLE.

Cornea and Sclera

THE PROBLEM OF CORNEAL HEMORRHAGE AND GLISTENING CORPUSCLES. Y. SHOJI and H. TANABE, Ann. d'ocul. **170:** 589 (July) 1933.

In 1875, Schmidt-Rimpler published a report on the type of apoplexy which Hirschberg calls hemorrhage of the cornea (Furchblutung der Hornhaut). Numerous reports have been made on this disease of which the clinical and histologic aspects are as follows: After operation or trauma of the globe, one often observes hemorrhage in the anterior chamber. If the absorption is slow one finds the cornea colored a yellow-green or dark brown. Histologically it is characterized by glistening corpuscles and pigmented granules in the corneal tissue. No cellular inflammatory infiltration is found. The details of two cases are then given.

If one examines the ophthalmologic reports concerning the relation between corneal hemorrhages and corpuscles and pigments, one can classify them into four groups: (1) clinically corneal hemorrhage and histologically glistening corpuscles and pigmented granules; (2) clinically corneal hemorrhage and histologically corpuscles but no pigment; (3) clinically corneal hemorrhage and histologically pigments but no corpuscles and (4) clinically corneal hemorrhage but histologically neither corpuscles nor pigments.

The first case reported belonged to the fourth group, but as the second did not seem to fit into any of the groups, a fifth group is suggested, namely, clinically hemorrhage of the anterior chamber but not of the cornea, and histologically glistening corpuscles in the corneal tissue.

S. H. McKee.

Traumatic Episcleral Abscess with Iritis Limited to the Adjacent Tissue. Pathogenesis of Sympathetic Ophthalmia. Van Lint, Arch. d'opht. **50:** 449 (July) 1933.

The expression "partial iritis" is used to indicate an inflammation limited to a segment of the iris. Van Lint cites a case in which a localized episcleral abscess resulted from an injury produced by the end of a piece of steel wire. In the corresponding sector of the iris an inflammatory reaction occurred and was restricted to this area. This partial iritis seemed to be due to the local episcleral abscess, but it was not noted until the abscess had healed. It would appear, therefore, to have been due to toxins which affected the neighboring iris because of its contiguity. In support of this contention reference is made to the partial dilatation of the neighboring iris when epinephrine hydrochloride is injected with procaine hydrochloride for surgical anesthesia. Van Lint believes that the pathogenesis of certain ocular disorders can be explained by the passage of toxins not only through scleral tissue but also through bone. In this way, he suggests, some cases of retrobulbar neuritis may be accounted for as due to sinus disease. The explanation is also applicable in certain dental conditions. In view of these observations he discusses two arguments which support the theory that sympathetic ophthalmia may be due to an infection which has passed through the neighboring tissues including the bony walls of the orbit. The first is that it may occur in a way similar to that in which the iritis was produced in the case cited, especially since tumor cells much larger than micro-organisms are seen to traverse the sclera. The second is based on the incomparably greater frequency of sympathetic ophthalmia in children.

S. B. Marlow.

Chemical Composition of the Proteins of the Cornea. Arlington C. Krause, Arch. f. Augenh. **107:** 453, 1933.

In a previous communication (*Am. J. Ophth.* **15:** 422, 1932) the author outlined the methods used for isolating the insoluble proteins, mucoid, gelatin from collagen, elastin and Descemet's membrane. In this communication he gives the results of further analyses of these substances.

He concludes that the mucoid of the substantia propria is a glycoprotein. It consists of mucoitinsulphuric acid, bound to a protein, which has not been identified. The mucoitin contains the acetyl-amino-mannose group. The mucoid of the vitreous is the only other mucoid in the eye which possesses the same chemical composition. This mucoid serves as the cement substance for the various layers of fibers of the cornea; its other functions are not as yet explained. It is not the same mucoid that exists in the sclera.

The gelatin found in the cornea comes from the collagen and is a typical gelatin. The elastin is similar in composition to that of the sclera.

Analysis of Descemet's membrane shows that it has a composition similar to that of the lens capsule.

F. H. ADLER.

DOES THE TREATMENT OF CONGENITAL SYPHILIS PREVENT THE DEVELOPMENT OF INTERSTITIAL KERATITIS? INTERSTITIAL KERATITIS AFTER INJURY. K. VOM HOFE, Klin. Monatsbl. f. Augenh. **90:** 492 (April) 1933.

In four cases reported by vom Hofe early treatment of congenital syphilis did not prevent later development of interstitial keratitis. Two of the patients, aged 11 and 13, had received antisyphilitic treatment in infancy; one, aged 12, had had such treatment about nine months prior to observation, and the fourth patient, aged 25, at the age of 14 years, after interstitial keratitis had developed. A recurrence followed in one eye two days after a small foreign body became embedded in the cornea. Numerous deep-seated vessels developed at the site of the foreign body, and parenchymatous cloudiness in its surroundings at a considerable distance. The cornea was fairly clear again a month later.

K. L. STOLL.

HYPOTONY PRODUCED BY PUNCTURE OF THE ANTERIOR CHAMBER AND ITS EFFECT ON PURULENT KERATITIS. T. SANO, Acta soc. ophth. jap. **36:** 116 (Sept.) 1932.

Sano studied the effect of paracentesis on purulent keratitis in rabbits and concludes: 1. Puncture of the anterior chamber reduces the severity of the inflammatory symptoms in purulent keratitis. 2. A definite therapeutic advantage is to be obtained by the puncture of the anterior chamber in purulent keratitis if it is done early or when the inflammation is mild.

A. KNAPP.

AN OPACITY RESEMBLING A SPIDER'S WEB SEEN ON THE POSTERIOR SURFACE OF THE CORNEA. K. HIROSE, Acta soc. ophth. jap. **36:** 121 (Oct.) 1932.

After a forceps delivery, an infant girl had suffered an injury to the right side of her head and to the right eye. The eye was swollen, and there was considerable discharge. This was quickly relieved and the eye apparently recovered except for a corneal opacity. When the child

was 15 years old, the following condition was discovered during a school inspection: There was a grayish-white opacity in the posterior layers of the cornea running from above and in, to down and out. In this opacity there were three parallel strands which, when examined with binocular magnifying glasses, proved to be situated posteriorly to the opacity and to bridge over the posterior surface of the cornea. These strands glistened like silk and appeared like fibers of a spider-web. The vision in both eyes was normal.

A. KNAPP.

General Diseases

THE HAND-SCHÜLLER-CHRISTIAN SYNDROME. S. PADOVANI, Arch. di ottal. **40:** 281 (June); 297 (July-Aug.) 1933.

The author reports a case presenting the characteristic picture of the Hand-Schüller-Christian syndrome. A boy, aged 7 years, had had polyuria, exophthalmos and anomalies of dentition at the age of 4 years. The legs were cyanotic; the few remaining teeth were irregular and abnormally large, and the skull was large and tender on pressure. Exophthalmos was more marked on the left side (23 mm., Hertel). Vision with correction was 1/2 in the right eye and 1/8 in the left; the fields and the fundi were normal except for slight dilatation of the retinal veins. About 7 liters of urine was passed daily. Roentgen examination showed defects in the skull, especially on the left side, where the upper, outer and lower orbital walls were almost entirely absent. The anterior and right posterior fontanels were not closed. The sella was large, and the anterior clinoid processes were eroded. As a result of the defect in the left orbital floor the left globe was displaced downward 4 mm. in comparison with the right one. The exophthalmos on this side may probably be explained as due to the pressure of the brain through the defective orbital wall.

S. R. GIFFORD.

OCULAR FINDINGS IN MONGOLIAN IMBECILES. W. VONTOBEL, Arch. f. Ophth. **130:** 325 (Aug.) 1933.

Twenty-five mongolian imbeciles (inmates of Swiss asylums) were studied to determine the changes in the lens and cornea. Up to the age of 7 years most of the patients had clear lenses. Then uncharacteristic opacities of the lens slowly developed. The opacities were of the caerule type, combined with opacities around the embryonic sutures. High degrees of hyperopia, strabismus (in fifteen of twenty-five cases) and blepharitis were also common among the mongolian imbeciles.

P. C. KRONFELD.

Glaucoma

THE RELATION BETWEEN ARTERIOSCLEROSIS AND GLAUCOMA. M. VELE, Ann. di ottal. e clin. ocul. **61:** 511 (July) 1933.

Vele recorded the blood pressure and Ambard's constant (the relation between the blood urea and the urinary urea) for fifty glaucomatous and fifty nonglaucomatous patients of the same age group. The average

of the systolic and of the diastolic blood pressure and of Ambard's constant was slightly higher in the glaucomatous group than in the non-glaucomatous group, Ambard's constant being highest in the cases of absolute glaucoma (0.125) and higher in cases of simple glaucoma than in those of the inflammatory type. A slight hypertension was the rule in glaucomatous patients.

S. R. GIFFORD.

CLINICAL VALUE OF TOLERANCE TESTS FOR DIAGNOSIS OF GLAUCOMA. R. STEIN, Med. Klin. **29:** 1235 (Sept. 8) 1933.

Stein points out that the functional disturbances in the intra-ocular vascular apparatus play an important part in the pathogenesis of glaucoma. Whereas the normal eye has the capacity to retain an equilibrium of pressure in spite of fluctuation in the general circulation, the glaucomatous eye or the eye that is predisposed to glaucoma has lost this capacity and reacts to changes of pressure in its vascular system with an increase in its internal pressure. This changed reaction is frequently already present at a time when the clinical symptoms of glaucoma are not yet clear. Thus there is a possibility of securing an early diagnosis of glaucoma by tolerance tests. The modification of the intra-ocular vascular apparatus in order to obtain a diagnostically valuable fluctuation in pressure can be produced by various methods. The author first describes and evaluates the test in which the injection of caffeine or drinking of strong coffee is used to obtain a noticeable increase of the intra-ocular pressure. Then he discusses the inhalation of amyl nitrite. This test may be modified by decreasing the number of drops to be inhaled from 5 to 3, and he shows that the diagnostic value of this test increases with this decreased dosage. Whereas the aforementioned tests are based only on the dilation of the vessels of the head, there are other tests which in addition to the increased inflow of blood also involve stasis. The latter can be produced by positional changes, particularly by lowering of the head or by constricting bandages. The author mentions a water test, the fluorescein test and the modification of the intra-ocular pressure by massage. He discusses Seidl's test (exclusion from light and exposure to light) and the influence of miotics and of mydriatics. He points out that the intra-ocular pressure does not remain constant but changes in the course of the day. In glaucomatous eyes these fluctuations are much greater than in the normal eye, and abnormally high fluctuations should therefore draw attention to the possibility of a glaucoma. Since none of the tests can be considered absolutely reliable, it is advisable always to employ several of them. After a successful operation the formerly positive reactions become negative, and thus the tests can be employed for the estimation of the results. Moreover, the tolerance tests indicate that the patient suffering from glaucoma should avoid everything that increases the dilatation of the vessels of the head, such as strong coffee, working in a bent position, wearing tight collars, resting in the horizontal position, and living in overheated or insufficiently ventilated rooms.

[J. A. M. A.]

Hygiene, Sociology, Education and History

THE CENTENARY OF THE STEREOSCOPE. GREEFF, Deutsche med. Wchnschr. **59:** 932 (July 16) 1933.

On June 21, 1833, the English physicist Charles Wheatstone presented before the Royal Society in London the first stereoscope. He was born in Gloucester, in 1802, and later became a maker of musical instruments. From 1823 to 1824, the *Annals of Philosophy* published his work on acoustic experimentation. One year after presenting the stereoscope, he was made professor of experimental physics at King's College. In 1837, he patented with Cooke an electric telegraph instrument. Wheatstone's stereoscope was of the mirror type in which the two different pictures of both eyes were formed into one plastic picture by means of two right-angled mirrors set at opposite ends.

Not until 1843 did David Brewster bring forth his prism or lens stereoscope. He was born in Scotland in 1781, and was first a pharmacist, later being appointed professor of physics at St. Andrews University in Edinburgh. In 1850, Brewster took his instrument to Paris where the optician Solail and his son-in-law, Dubosq, recognized its importance and created a popular appeal.

In 1857, von Helmholtz with his telestereoscope discovered the fact that pupillary distance was the guide to depth perception.

The author calls attention to the many instruments which have been evolved from these sources: namely, the stereoscopic camera, the binocular microscope and the periscope. The advantage of stereoscopic roentgenograms is noted. A bibliography accompanies the article.

L. L. MAYER.

Injuries

DAMAGE TO THE EYES BY HAIR DYE. W. BAB, Deutsche med. Wchnschr. **59:** 1041 (July 7) 1933.

Six cases of severe reactions following the use of henna hair dye, seen during a period of four years, are reported. In spite of the fact that some authorities have referred to this plant dye as harmless, and also that the textbooks on ophthalmology fail to record the harmful effects, the author warns of the severe reactions which may result from its use. The symptoms of edema of the lids and conjunctiva, photophobia, blepharospasm and at times concurrent infection are based on an allergic phenomenon. Intense pain which does not respond to the usual medicaments causes great discomfort and loss of sleep. The symptoms begin from twelve to twenty-four hours after the application of the dye. Treatment must be continued from twelve days to as long as seven weeks in some cases.

L. L. MAYER.

LESIONS OF THE CONTENTS OF THE ORBIT DURING ENDONASAL CURETTING OF THE ETHMOID CAVITY: REPORT OF TWO CASES. H. F. ROESE, Klin. Monatsbl. f. Augenh. **91:** 95 (July) 1933.

Two cases of curettage of the ethmoid cavity are reported, during which the lamina papyracea was perforated incidentally.

In the first case, that of a man, aged 42, a considerable hematoma developed immediately in the orbit, coupled with swelling of the lids and double vision, caused by dehiscence of the internal rectus muscle. Infection of the hematoma led to the formation of thick scar tissue around this muscle, which frustrated an attempt to advance it.

The second patient, a woman, aged 30, felt severe pain in her left eye during the curettage of the left ethmoid cavity. Her eye was blind since that day; enophthalmos, slight ptosis and paralysis of the entire oculomotor nerve were present on the next day. No hemorrhage occurred in the nose or in the orbit. The fundus was free from pathologic changes, until genuine optic atrophy revealed the presence of a rupture of the optic nerve three weeks later. The enophthalmos was explained as the result of a hernia-like escape of some of the contents of the orbit through the perforated lamina cribrosa into the ethmoid cavity. The conjunctiva and cornea were asensitive, indicating dehiscence of the first branch of the trigeminus nerve.

K. L. STOLL.

Instruments

A NEW ANTERIOR SYNECHIA KNIFE. C. V. KRISHNASWAMI, Brit. J. Ophth. **17:** 419 (July) 1933.

The instrument consists of a short narrow blade with blunt end and sharpened edges down to within 3 mm. of the tip. After incision with a keratome, the instrument is introduced into the anterior chamber until the point is well past the site of the adhesion. Either by depressing the handle or by a series of sawing movements, the adhesion is severed. The instrument is depicted.

W. ZENTMAYER.

A NEW SLIT LAMP. A. G. MACKIE, Brit. J. Ophth. **17:** 554 (Sept.) 1933.

Mackie believes that the slit lamp would be more generally used if it were not for its many complicated adjustments, which necessarily increase the cost of the apparatus.

By improvement in design some of these accessories can be rendered unnecessary and expense saved. With these ends in view the author has designed a new model.

W. ZENTMAYER.

Lacrimal Apparatus

RESULTS OF INTRANASAL DACROCYSTORHINOSTOMY. L. M. J. HENRY, Brit. J. Ophth. **17:** 550 (Sept.) 1933.

The operations were performed by Fraser at the Royal Infirmary, Edinburgh. The investigation was undertaken in order to test the results of the West operation. The results were studied from three aspects: clinical, i. e., the patient's statement regarding her or his condition; the functional efficiency of the new passage from the conjunctiva to the nose, as tested by the fluorescein test; bacteriology of the conjunctiva, to determine whether the operation could be used for "sterili-

zation" of the conjunctival sac before extraction of the cataract, or before other intra-ocular procedures.

The summary of the results shows that twenty-three cases (four after bilateral operation) were investigated. A perfect clinical result was obtained in 78 per cent (compared with 73 per cent in 1925 [Fraser, J. S.: *J. Laryng. & Otol.* **40:** 725, 1925]). The fluorescein test apparently may show obstruction in cases in which there is no epiphora and therefore is not of great practical importance. Sterilization of the conjunctival sac as regards streptococci and pneumococci was obtained in 82 per cent.

W. ZENTMAYER.

CALCIFICATION OF THE TEAR GLAND AS SEEN IN THE ROENTGENOGRAM: REPORT OF A CASE. J. IGERSHEIMER, Klin. Monatsbl. f. Augenh. **90:** 473 (April) 1933.

A man, aged 39, was seen in November 1930, with a receding swelling of the left side of the face and an exophthalmos of 7.5 mm. A tender, resistant mass could be felt in the area of the left orbital lacrimal gland, and the preauricular glands were slightly enlarged. The fundus was normal aside from slight distention of the retinal veins, slight hyperemia of the disk and blurring of its margin; vision was normal. The roentgenogram showed the shadow of a mulberry-shaped calcification at the left temporal margin of the orbit. The pain and irritation disappeared, and the exophthalmos receded to 5 mm. within a few weeks and was only 3 mm. in October, 1932. Pictures of the patient taken from 1914 on gave evidence of slight exophthalmos. After ruling out a tumor of the lacrimal gland and exostoses or phlebolites of the orbit, Igersheimer diagnosed tuberculous calcification of the lacrimal gland. An acute intervening inflammation, in his opinion, had caused the transient increase of the exophthalmos. This view was corroborated by the roentgenologic diagnosis of Groedel and Lossen.

K. L. STOLL.

Lens

INTRACAPSULAR EXTRACTION OF THE CRYSTALLINE LENS CONTAINING A FOREIGN BODY. B. CHANCE, Am. J. Ophth. **16:** 597 (July) 1933.

The author discusses the problems involved in wounds of the eye in which foreign bodies have lodged in the lens. Three cases in which he successfully extracted the lens in its capsule with the contained foreign body are reported. He cites the advantages of this method over other procedures.

W. S. REESE.

DIABETIC CATARACT. LT.-COL. E. O'G. KIRWAN, Brit. J. Ophth. **17:** 346 (June) 1933.

As diabetes mellitus is probably more frequent in Bengal than in any other part of the world one expects to see there a large number of cases of diabetic cataract. Kirwan's experience coincides with that of Foster Moore: The incidence of cataract is greater in persons with diabetes than in nondiabetic persons of the same age, and as a result of diabetes lens changes appear at a relatively earlier age and tend to mature more rapidly. In early life there is an undoubted association of

cataract with diabetes. This cataract occurs when the disease is severe and develops rapidly, in both sexes. The opacities occur first in the posterior cortical layers and extend into the anterior cortical layers; they are situated immediately beneath the capsule.

Four cases of diabetic cataract with definite morphologic features are reported. The report is illustrated. W. ZENTMAYER.

THE IDENTITY OF CATARACT FORMATIONS. J. FOSTER, Brit. J. Ophth. **17:** 408 (July) 1933.

The object of the author in this paper is, by pruning detail and stressing practical points, to give a working classification of the types of cataract and their appearance as seen with the slit lamp. Nothing new is offered. The article is illustrated. W. ZENTMAYER.

LOSS OF FLUORESCENCE OF LENSES IN ULTRAVIOLET RAYS. C. KOCH and F. P. FISCHER, Arch. f. Augenh. **107:** 440, 1933.

In 1926, Thiel (*Ztschr. f. Augenh.* **58:** 86, 1926) found that lenses with developing cataracts began to lose their fluorescence in ultraviolet rays and that the loss of fluorescence more or less paralleled the destruction of the lens fibers.

The authors set themselves the task of determining what physical or chemical changes in the lens were responsible for this loss of fluorescence. They examined whole animal lenses and lenses cut in thin sections according to the method of Vogt (*Klin. Monatsbl. f. Augenh.* **51:** 129, 1913) with the rays from a filtered arc. In such rays the lenses fluoresced to a gray-blue. These lenses were then treated with different acid and alkaline solutions which were known to bring about swelling of lens fibers. Boiling and freezing and mechanical precipitation of the lens protein were also investigated. None of these methods succeeded in causing any change in the fluorescence of the lenses. From this the authors are able to say that the change in fluorescence in lenses containing cataracts is not due in any way to changes in the water content of the lenses.

A series of experiments were then undertaken which proved that the loss of fluorescence is not due to the state of the proteins in the lens. Breaking these down with pepsin or trypsin in no way changed the fluorescence. Similarly, the *alpha* or *beta* crystallin content of the lenses made no difference in the degree of fluorescence.

However, when aqueous humor was dropped on the lens sections the fluorescence vanished. The same was true for blood serum containing hemoglobin and for an ultrafiltrate of blood serum. Artificial aqueous humor did not change the fluorescence.

The authors were unable to determine just what physical or chemical properties of the aqueous or the serum were responsible for this effect. Fluorescence is an emission of light energy without any elevation in temperature, and according to the quantum theory this is a process bound up with the atom which occurs only when the atom takes up a definite quantum of energy. Something must be present in the aqueous or the serum which prevents the lens atoms from taking up the light energy, but just what this is is not known.

A point of some importance which the authors stress is that these experiments tend to prove that during life, when the lens is known to fluoresce, the aqueous humor must be prevented from reaching the lens fibers. It has been generally believed, and many experiments in recent years support this idea, that throughout life the capsule of the lens is permeable to the aqueous humor, and that the lens derives its nutrition from the aqueous which reaches it in this manner. The authors point out that their present experiments make this hypothesis untenable.

F. H. ADLER.

DYSTROPHIA EPITHELIALIS LENTIS ADIPOSA. REPORT OF A CASE. A. VON SZILY, Klin. Monatsbl. f. Augenh. **90**: 607 (May) 1933.

A man, aged 59, was first seen in January 1931, a few months after his hitherto good eyes began failing rapidly. A slight decrease of vision had been noticed for a few years. Both eyes were normal with the exception of an opacity in the anterior pole of each lens. The shape of the opacity in the right eye was triangular and that of the opacity in the left was horizontally oval. Short, fine radiating extensions and numerous flat or needle-shaped cholesterol crystals of various colors were observed in both opacities. The opacities grew more intense and the embedded cholesterol crystals more numerous during ten months of observation. The right lens was extracted and perfect vision obtained. Histologic examination of the lens showed slight indentation at the anterior pole. This indentation was filled by proliferating capsular epithelium. Large numbers of fat globules and cholesterol crystals were interspersed between the degenerated cells of this area, while the surrounding portions of the lens were perfectly clear. The patient's metabolic rate was 2.3 per cent below normal; the cholesterol and lipoid contents of the blood varied; a slight degree of cholesteremia was present, 214 mg. per hundred cubic centimeters being found in the serum. Von Szily is inclined to exclude the congenital form of anterior polar cataract and, discussing the rôle played by fatty degeneration in cataractous processes, expresses the opinion that the lipoid substances found in cataracts are not identical with those formed in autolysis. He denies the existence of an analogy between intravital cataractous and postmortem autolytic degeneration. The fatty infiltration, or lipoidosis, found and described prompted him to consider the condition as a dystrophy of the epithelium of the lens. Although the hypercholesteremia was slight, nevertheless the constant exposure of the eyes to light, and perhaps hormonal processes, may have accelerated the development of the opacities.

K. L. STOLL.

Lids

GUMMA OF THE LOWER LID. RYSS-ZALKIND, Sovet. vestnik oftal. **3**: 55, 1933.

A patient, aged 21, revealed on examination a firm swelling the size of a walnut at the inner angle of the eye. In two days the swelling broke down. The necrotic matter obtained from it was examined for spirochetes and tubercle bacilli, but none were found. Histologic exam-

ination showed connective tissue in a state of incomplete necrosis; in the walls of the blood vessels there were marked necrotic changes. The Wassermann reaction was positive. An ulcer with undermined, infiltrated edges and a deep fatty base formed in the place of the former swelling. After five injections of neoarsphenamine the ulcer healed, and a fine cicatrix formed.

The diagnosis of gumma was made because of the appearance of the ulcer, the histologic observations, the absence of swelling of the regional glands and the absence of spirochetes in the lesion.

O. SITCHEVSKA.

Methods of Examination

OPHTHALMOLOGIC INTERPRETATION OF RARE FINDINGS WITH ROENTGENOGRAMS OF THE SKULL: REPORT OF CASES. OSKAR THIES, Klin. Monatsbl. f. Augenh. **91**: 38 (July) 1933.

The author refers to the ophthalmoroentgenologic atlas of Rudolf Thiel, and expresses his opinion that the authorities will request roentgen diagnoses from the oculist in the future, as they are requesting them from the surgeon at the present time. A number of cases are reported to maintain this view.

The first case is mentioned to show that disease of the nasal cavities was the only cause of retrobulbar neuritis at a time when the rhinologic findings were still doubtful.

In the second patient, a man, aged 48, protrusion and temporal deviation of one eye resulted from an accident. The vision was found greatly reduced when he recovered consciousness on the fourth day after the accident. The roentgenogram established the diagnosis for treatment and for compensation proceedings.

In the third and fourth cases the roentgenograms not only gave evidence of the exact size and location of intracranial tumors, but allowed definite prognostic conclusions, which were impossible by prior encephalography.

The fifth case, that of a man, aged 78, with myopia, marginal excavation of both disks and intra-ocular tension of from 26 to 28 mm., was cleared up diagnostically after the roentgenogram showed calcification of the internal carotid artery extending from the sella turcica to the anterior clinoid process. It was a case of glaucoma without increased tension due to severe cerebral arteriosclerosis.

Characteristic degeneration of tertiary syphilis of the clinoid processes was found by the roentgenogram in two further cases, in which only minor ocular symptoms were present. Diagnostic doubt may arise between tertiary syphilis and osteosarcomatous processes, until the porous appearance of the bones, typical for the former disease, has been observed.

Sarcomatosis of the base of the skull with a metastasis in the right petrous bone was found in the last case as a sequela to sarcoma of the femur. Convergent squint of one eye was the only ocular symptom. Exact anamnesis and a Wassermann test of the lumbar fluid remain essential in these cases.

K. L. STOLL.

THE IMPORTANCE AND TECHNIC OF THE EXAMINATION OF THE LIGHT SENSE BY THE MEDICAL ADVISOR. E. ENGELKING, Med. Wchnschr. **80:** 923 (July 16) 1933.

The unprecedented speed of modern vehicles has created an importance, heretofore lacking, of the light sense of the eye. Together with this increase in speed are the rapid changes in the illumination of one's surroundings. To those in charge of the locomotion of these rapid vehicles, the measurement of the light sense is indeed a safety factor. Adaptation and its study comprise a costly, tedious examination relegated to a specialist in ophthalmology. The author recommends methods to be used by the medical advisor which are practical, efficient and not expensive. The scotopticometer of Moller and Edmund is a small, cheap means of examination. If the patient is able to read the three darkest characters in from two to four minutes, he is said to have a normal adaptation ability. Another method is to have both examiner and patient come from the bright sunlight into a dark room. The ability of the patient to recognize the figures on a watch dial in comparison to the time it takes the normal examiner is a good indication of the light sense. Patients with gross deviation from the normal should be sent to the ophthalmologist for a more complete study.

L. L. MAYER.

Neurology

KAYSER-FLEISCHER RING IN WILSON'S DISEASE. F. L. DUNNAVAN and M. PAUL MOTTO, Am. J. Ophth. **16:** 571 (July) 1933.

The literature on Wilson's disease is reviewed, and a case reported of a white girl, aged 14, first seen on March 4, 1932. She complained of lack of appetite, retarded and laborious speech and inability to walk or sleep well. She had been treated for encephalitis. Since her eighth year she had displayed intermittent tremor of the hands as well as some difficulty in eating, and inability to talk plainly. Her walking became decidedly worse, and she began to lose control of the finer movements of her hands and arms so that she was practically bedridden. Examination revealed decreased dulness of the liver and athetoid movements of the hands and fingers with spasticity. The gait was of a staccato, shuffling, spastic type. Ocular examination revealed a circular dull olive-green band about 2.5 mm. broad around the periphery of each cornea. The biomicroscope showed that this pigmentation was confined to Descemet's membrane and consisted of brownish granules. The eyes were otherwise normal. The patient died on May 28, 1933, and permission for autopsy was not secured. The authors point out that the Kayser-Fleischer ring is an important symptom of Wilson's disease, and is of special significance from the standpoint of differential diagnosis.

W. S. REESE.

HYSTERIC AMBLYOPIA. F. C. CORDES and W. D. HORNER, Am. J. Ophth. **16:** 592 (July) 1933.

The cause, symptomatology and treatment of hysteric amblyopia are discussed, and three cases are reported. The patient in case 1 was a graduate nurse, aged 24, who married at the age of 21 because of her desire for a child. One year later her pregnancy was terminated at eight months, the child living only six hours. Delivery by forceps resulted in a marked third degree tear, and to make matters worse her husband deserted her at this time. One month before admission her vision failed rapidly, and when examined she claimed only perception of light in each eye. She was told that a general anesthetic would cause the visual impulses to start flowing again, and her vision would return to normal. This prophecy proved correct. The patient in case 2 was a man, aged 36, whose occupation was evidently not to his liking. Loss of vision developed after an examination in which drops were used in his eyes, and he was subjected to an exhaustive physical examination. This was made because of claims for disability insurance. The patient was told that his spinal fluid was under pressure, and his vision was restored after an unsuccessful lumbar puncture. The patient in case 3, a woman, aged 36, had had a number of abdominal operations and loss of vision in one eye following the use of drops. As in case 2, her vision was restored by lumbar puncture. The authors comment on the manner in which these cases arise and the therapeutic measures necessary.

W. S. REESE.

THE OCULAR DISTURBANCES IN EPIDEMIC ENCEPHALITIS. C. P CLARK, Am. J. Ophth. **16:** 606 (July) 1933.

The author discusses the ocular manifestations in epidemic encephalitis during the acute and chronic stages. In the former are seen muscle palsies (fugacious in character), ptosis and pupillary changes. Changes in the fundus appear to be uncommon. During the chronic stage there occur ptosis; paralysis, especially of accommodation; oculogyric crises; disturbances of disjunctive movements; nystagmus; disturbances of the orbicularis; changes in the pupils and the fundus, and ocular manifestations of a psychic character. The author cites several illustrative cases, and briefly discusses the treatment.

W. S. REESE.

DISTORTION OF VISUAL FIELDS IN CASES OF BRAIN TUMOR: FIELD DEFECTS AND HALLUCINATIONS PRODUCED BY TUMORS OF OCCIPITAL LOBE. G. HORRAX and T. J. PUTNAM, Brain **55:** 499 (Dec.) 1932.

According to Horrax and Putnam, intracranial tumor confined to the occipital lobe is comparatively rare; it occurred in only 40 of 1,881 verified cases of intracranial growths. In 73.6 per cent of patients with a tumor confined to or chiefly compressing the occipital lobe the fields of vision showed a contralateral homonymous hemianopia in which the central fibers remain unaffected. This was particularly true of meningioma situated at the posterior end of the occipital lobe. An upper quadrantal homonymous defect of the visual fields was not disclosed in any

case of the series, and its occurrence in the presence of a strictly occipital tumor is therefore regarded as rare. Visual hallucinations occurred in 6 patients (15 per cent) before operation for removal of the tumor. Careful examination of the hallucinations supports the idea that complex "formed" images are not provoked by occipital tumors. Differential diagnosis between a temporal and an occipital tumor may be impossible without ventriculography. The high incidence of spared central fibers and the rarity of upper quadrantal defects with occipital growths are helpful, since bisection of the macula and upper quadrantanopia are frequent with temporal tumors. Other differential features are the relatively greater tendency to the true quadrantic form (bounded by the horizontal meridian) in defects due to lesions of the temporal lobe, and their greater incongruity as compared with those due to tumors of the occipital lobe.

[J. A. M. A.]

HERPES ZOSTER OPHTHALMICUS. J. H. DOGGART, Brit. J. Ophth. **17:** 513 (Sept.) 1933.

The author first describes the general features of herpes zoster ophthalmicus, and then takes up the ocular complications, which he states are keratitis, iridocyclitis, ocular palsies and optic neuritis. Impaired corneal sensation, scleritis and patches of atrophy in the iris are mentioned as sequelae.

Four illustrative cases are cited. In the first the ocular symptoms consisted of severe iridocyclitis with dense opacities in the deeper layers of the cornea, increased tension, slate-colored scleral patches and blindness.

In the second case there were iridocyclitis and palsies of the sixth nerve. Both complications subsided, and ultimate visual acuity was 6/9.

In the third case the only ocular complication was mild keratitis.

The chief point of interest in the fourth case was that the blebs appeared on the cornea before typical zoster keratitis arose.

W. ZENTMAYER.

OCULAR SYMPTOMS IN INFECTION WITH SPIROCHAETA ICTEROHAEMORRHAGIAE. R. T. BRANBERGEN, Arch. d'opht. **50:** 485 (July) 1933.

Branbergen cites three cases of infection with Spirochaeta icterohaemorrhagiae in which optic neuritis was observed. The condition has been referred to by other reporters, one of whom considers it evidence of involvement of the central nervous system. Branbergen believes that the recognition of this condition in such cases will be of help in the differential diagnosis between the meningeal form of the disease and meningitis. Its occurrence is in accord with the opinion expressed by Widal that there is an affinity between this organism and nervous tissue.

S. B. MARLOW.

RELATION BETWEEN INTRACRANIAL RACEMOSE ANGIOMA OR CIRSOID ANEURYSM AND TORTUOSITY OF THE RETINAL ARTERIES. K. WESSELY, Klin. Monatsbl. f. Augenh. **90:** 95 (Jan.) 1933.

A girl, aged 19, had had a severe intracranial disorder with loss of consciousness for four weeks in the second year of her life. Ever since

that time she had suffered from epileptiform attacks and headaches. Left-sided nonpulsating exophthalmos developed during the year preceding observation, and the conjunctival and scleral vessels, the left temporal artery and the retinal vessels in both eyes became tortuous. Systolic bruit, not perceived by the patient, could be heard by auscultation all over her head. The sulci of the meningeal arteries, commonly barely visible in the roentgenogram, could be observed as broad, tortuous and frequently anastomosing clear bands in the bone; these were interpreted as the pictures of abnormally wide, tortuous arteries, forming frequent convolutions. A similar condition was found in other patients presenting uncomplicated tortuosity of the vessels in the fundus. These findings suggest that an abnormal course of the retinal vessels may occur physiologically with a similar formation of the intracranial vessels.

K. L. STOLL.

Ocular Muscles

PARALYTIC ALTERNATING STRABISMUS OF ? ORIGIN. E. A. SEALE, Brit. J. Ophth. **17:** 415 (July) 1933.

Though "alternating" is usually applied to concomitant squints, the action in the case reported is a striking feature. A girl, aged 5½ years, had an ocular condition which was first noticed when she was a few months old, following a slight accident.

Vision in each eye was 6/6. Each eye could fix equally well. When the right eye fixed, the left eye looked in and the left lid drooped. When the left eye fixed, the right eye turned up and in, the eye being then widely opened. There were a facial paralysis of the left side extending to the tongue and the lips, a weakness of the levator of the left lid and a slight deafness on the left side. The possible location of the lesion is discussed. It is suggested that the nucleus of the sixth nerve on the right side is the chief seat of the defect. The article is illustrated.

W. ZENTMAYER.

STRABISMUS-AMBLYOPIA AND ITS TREATMENT. REPORT OF CASES. W. HERZAU, Klin. Monatsbl. f. Augenh. **90:** 612 (May) 1933.

A man and his three daughters showed unilateral amblyopia. Only the father and one daughter had strabismus. The father's amblyopia was functional, the vision of his weak eye increasing from 1/12 to 6/18 after he had lost the normal eye through an accident. In two of the daughters, Herzau admits, slight strabismus may have been present but hidden by eccentric fixation. A predisposition to the development of functional amblyopia may be inherited.

Several tables illustrate the effect of unilateral occlusion bandages on thirty-seven children ranging in age from 2 to 7 years. The bandages were applied for periods of from seven to eleven weeks.

Herzau arrived at the following conclusions: Functional amblyopia is the rule and congenital amblyopia the exception in strabismic persons. Combinations of both types occur. The onset of strabismus in earliest childhood and possibly major deviation may influence the development of amblyopia. Very young children respond the most promptly

to occlusion bandages, but they are prone to suffer recurrences, and occasionally amblyopia in the occluded, stronger eye. Good final results will be obtained in older children by longer periods of occlusion. After elimination of the strabismus binocular stereoscopic fusion resulted frequently but binocular depth perception rarely. In a number of children fusion was lacking after elimination of the deviation. Vision, however, did not decrease after the formerly amblyopic eye had assumed fixation temporarily.

K. L. STOLL.

SO-CALLED DIVERGENCE PARALYSIS. H. F. ROESE, Klin. Monatsbl. f. Augenh. **90:** 617 (May) 1933.

Divergence paralysis (after Bielschowsky) is characterized as follows: 1. There is suddenly an observance of double images of all objects beyond a distance of from 25 to 50 cm. 2. The convergence of the eyes causing the double images remains unaltered or the discrepancy may even decrease on turning the eyes to either side. 3. The double images fuse on the approach of the fixation object, frequently at the usual working distance, and binocular vision persists even on lateral motion of the eyes. 4. Adducing prisms produce binocular vision in the entire visual field. 5. Gross contractions of the visual field are not evident. 6. The angle of squint remains unchanged, especially in cases with minor aberrations.

A man, aged 65, acquired these symptoms suddenly while riding on a train in October 1931. The disturbance disappeared to some extent, but returned in January 1933. The Wassermann reaction and the results of the neurologic examination were negative.

The question whether genuine divergence paralysis exists can be settled only after anatomic proof has been furnished that typical divergence paralysis may change to typical simple paresis of the abducens muscle. This would be simultaneous proof for the existence of a special center for innervation of divergence, the existence of which seems to be suggested by the case described.

K. L. STOLL.

Orbit, Eyeball and Accessory Sinuses

CLINICAL OBSERVATIONS ON THE INTRAOCULAR TENSION OF OPIUM HABITUÉS IN FORMOSA. KANJI KANDA and KAZEN SO, Brit. J. Ophth. **17:** 354 (June) 1933.

From their observations of opium habitués in Formosa the authors reach the following conclusions:

Although opium habitués for the most part have contracted pupils (in the authors' cases the average was 2.8 mm. diameter) the intraocular tension is either normal or somewhat higher than normal, the average values being 22.59 mm. of mercury with the Schiötz tonometer, the 5.5 Gm. weight being used.

Although the opium habitués are in a parasympathetic-tonic or vagotonic condition (and there is the fact that most patients with glaucoma are said to be in this condition, and conversely those who are in this condition may be more susceptible to glaucoma) the intra-ocular tension is, in general, normal, though in a few habitués it is a little higher or even lower than the normal limit of intra-ocular tension.

The curves of the values of the intra-ocular tension of the opium habitués are quite irregular in themselves and have no feature in common. This fact may be looked on as characteristic of the addiction. The curves do not seem to be much influenced by the withdrawal of opium or by the treatment of the habit.

The blood pressure of the opium habitués is, on the whole, hypotonic, the systolic pressure being 113 millimeters of mercury, and the diastolic 75, on an average. However, the intra-ocular tension is normal or even a little higher than normal, so that there can be found no definite parallel between it and the blood pressure.

A bibliography is furnished.

W. ZENTMAYER.

AN ORBITAL HEMORRHAGE WITH VOMITING. Y. TUTUI, Acta soc. ophth. jap. **36:** 115 (Sept.) 1932.

A boy of 15 years received a blow on his eyebrow, which resulted in an orbital hemorrhage, followed by repeated vomiting for two days. Examination of the vegetative nervous system gave the following results: The reactions to epinephrine, pilocarpine and atropine and the Aschner phenomenon were positive. This showed a sensitiveness of the entire vegetative nervous system and a mixed form of sympathicotonia and vagotonia. The vomiting is explained by the trigeminal vagus stimulation in the form of an oculogastric reflex in which vagotonia played a certain rôle.

In another case of prolonged vomiting after exenteration of the eyeball there was a large effusion of blood into the scleral cup. The vomiting was produced by pressure on the nerve stumps of the trigeminal nerve in the sclera and acted as an oculogastric reflex.

A. KNAPP.

Pharmacology

AN EXPERIMENTAL STUDY OF A NEW MIOTIC (CARBAMINOCHOLINE). M. APPLEMANS, Compt. rend. Soc. de biol. **115:** 1259, 1933.

In living rabbits, the instillation into the conjunctival sac of carbaminocholine hydrochloride in 0.1 per cent solution contracts the pupil. With the use of a 0.0001 per cent solution, a subconjunctival injection is effective in two minutes, and an injection into the anterior chamber produces instantaneous miosis lasting for an hour. The activity of this derivative of choline is extraordinary, but transitory.

J. E. LEBENSOHN.

The Pupil

THE PUPILLOMOTOR EFFECTS OF SOME OF THE MODERN LOCAL ANESTHETICS. K. VELHAGEN JR., Arch. f. Augenh. **108:** 308, 1933.

This is a report of the action of some of the newer local anesthetics on isolated strips of the iris. Five anesthetics were investigated: pantocaine (I. G. Dyes) (*p*-n-butylaminobenzoyl of dimethylaminoethanol hydrochloride); larocaine (Hoffmann La Roche) (*p*-aminobenzoyl-2, 2-dimethyl-3-diethylamino-propanol hydrochloride); diocaine

(Ciba) (*di*-allyloxyethyl-*di*-phenylamidine hydrochloride); *d*-iso-cocaine with sodium tartrate; holocaine (phenacaine).

Tests with the Isolated Dilator Pupillae.—The preparations were considered sufficiently sensitive for use if the iris tissue reacted to epinephrine in 1:4,000,000 dilution. When an active contraction had been produced by epinephrine this was washed off and the chosen anesthetic added in increasing concentration. None of the five anesthetics tested was nearly so effective as cocaine.

Tests with the Isolated Sphincter Pupillae.—For this purpose the sensitivity of the preparation was tested by the addition of carbaminoylcholine, which causes marked contraction of the isolated sphincter. All of the substances tested had about the same power to inhibit the contractions due to carbaminoylcholine as cocaine.

From a practical point these experiments show that a dilatation of the pupil may occur in patients after the exhibition of the newer anesthetics if these are given in sufficiently strong amounts and act for a sufficiently long period of time. If one wishes to be sure that there is no dilatation of the pupil after administering any of these drugs it is advisable to instil a miotic.

F. H. ADLER.

Physiology

THE VISIBILITY OF ULTRAVIOLET RAYS. J. SAIDMAN, J. A. M. A. **101:** 535 (Aug. 12) 1933.

Ultraviolet rays have been regarded as invisible to the human eye. That is not absolutely true; at any rate, the visibility varies with the age of the person. Saidman has made a filter that absorbs completely the rays of the visible spectrum and also the beginning portion of the ultraviolet rays, while preserving good transparency up to 3,100 angstroms. With this filter he studied the visibility of the ultraviolet rays. He found that the visibility of the 3,130 region, though constant in youth, disappears between the ages 34 and 43, or thereabouts. Of the seventy-four persons who saw the ultraviolet rays with both eyes, fifty-two stated that the impression was alike for the two eyes, and twenty-two saw much better with one eye than with the other. The human crystalline lens is therefore not so opaque as was supposed. The disappearance of the visibility of ultraviolet rays up to 3,130 angstroms would indicate an aging of the crystalline lens, and the procedure described might, the author states, be used to detect the beginning of ocular sclerosis.

W. ZENTMAYER.

THE PRIMARY POSITION OF THE EYES. N. A. STUTTERHEIM, Brit. J. Ophth. **17:** 394 (July) 1933.

The purpose of this paper is to show that the conception of the primary position of the eyes, as described in textbooks, is wrong. As against the traditional arbitrary method of defining a "primary position" of the bi-unial eye, the real primary position is the anatomic position of the organ.

This anatomic position is one of divergence of the visual axes. The globes are firmly fixed in their orbital beds by connective tissue, liga-

ments and strands which all contain a strong quota of elastic elements. The result of this is a stable equilibrium and an elasticity exercising permanent influence on motions and positions of the organ.

Phoria is a name for all secondary positions. These are characterized by variation and instability.

The extrinsic muscles act as tensors between the primary position and the edge of the fixation field. They act by contraction in the direction away from the primary position. In the direction toward the primary position they act by inhibition only. There is a system of reciprocal action between extrinsic muscular power and the power of elasticity of the primary position. The terms "muscle balance" and "muscle imbalance" are based on a wrong conception of anatomic and physiologic facts so far as they relate to the eye.

W. ZENTMAYER.

VITAMIN A IN THE RETINA. G. WALD, Nature **132:** 316 (Aug. 26) 1933.

Vitamin A is found not only in the intact retinas of frogs, sheep, pigs and cattle, but also in the pigment layers of the choroid and in solutions of visual purple. The specific chemical test for vitamin A—the antimony trichloride coloration—exhibits spectroscopically a strong, sharp band at 6,200 angstroms. The smoothness of the curves of absorption indicates that no other carotenoids are present in the extracts.

The oil derived from an extract of ox retinas gave a color intensity corresponding to 0.2 per cent vitamin A. One milligram of this oil was sufficient to cure rats displaying symptoms of avitaminosis. It is suggested that a diminution of this vitamin in the eye may be a factor in night blindness.

J. E. LEBENSOHN.

RESEARCHES ON THE LYSOZYM OF TEARS. L. VENCO, Rassegna ital. d'ottal. **2:** 519 (July-Aug.) 1933.

Studies of the lysozym present in human tears, normal and pathologic, confirm the belief that a high lytic potency characterizes this principle. Using as an indicator cultures of Micrococcus lysodeikticus of Fleming, Venco demonstrated that certain diseases of the ocular apparatus and even certain general morbid states reduce more or less the lysozymic index of tears. In some experiments made to test the bacteriolytic power of lysozym against micro-organisms, he found evidence of marked sensitiveness on the part of the majority of saprophytes, while pathogenic bacteria did not seem to resent, to any degree, its lytic action.

V. R. SYRACUSE.

THE BLOOD PRESSURE IN THE INTRA-OCULAR BLOOD VESSELS. W. WEGNER, Arch. f. Augenh. **108:** 313, 1933.

Most of the measurements of the blood pressure in the intra-ocular blood vessels have been made on the retinal system. Those who have made comparative measurements on both the retinal and the choroidal system usually agree that the pressures in these two systems are practically identical. It is also generally recognized that no comparisons

are permissible between measurements made in the intra-ocular blood vessels and those made in the extra-ocular vessels.

The present paper concerns the report of a clinical observation which led Wegner to believe that the pressure in the retinal system differs considerably from that in the choroidal system.

The case is that of a 43 year old woman from a family with a history of glaucoma, who had had juvenile glaucoma for fifteen years. In spite of miotics the condition gradually became worse. There was a marked glaucomatous cup, and pulsating choroidal as well as retinal arteries could be seen in each eye. The choroidal vessels were about the same size as the main retinal arteries at the edge of the papilla. An area of choroidal atrophy surrounded each papilla. The tension was 49 mm. of mercury in each eye. Vision in the right eye was limited to perception of hand movements; in the left eye to counting of fingers.

After instillations of "Links-Glaucosan" the pupil dilated and the tension fell. Before the final fall in pressure, however, the tension rose within the first fifteen minutes to 56 mm. of mercury in each eye, without any change in the pulsations in the vessels. Thirty minutes later the tension was 53 mm. of mercury; forty-five minutes later the tension in the right eye was 53 mm., and that in the left, 50 mm.; sixty minutes later the tension in the right eye was 56 mm., and that in the left, 44 mm. All this time the pulsations in both retinal and choroid systems continued. After seventy-five minutes the pressure in the right eye was 45 mm., and that in the left, 39 mm. At this time the pulsations in the right eye were unchanged, whereas in the left eye the choroid pulse had stopped, with no change in the retinal pulse. After two hours the pressure in the right eye was 45 mm., and that in the left, 38 mm. Again the pulsations in the right eye remained unchanged, whereas in the left only the pulsation in the retinal system showed. Five hours later the pressure in the right eye was 45 mm., and that in the left, 18 mm. At this time the pulsations in the right eye were as previously, and no pulsation could be observed in the left eye, either in the retinal or in the choroidal system. Further, there was no venous pulsation in the left eye. This condition remained about the same on the next day, with the exception that a venous pulse could be seen in the left eye.

Pressure applied to the left eye with the Bailliart dynamometer now showed a distinct arterial pulse when the intra-ocular pressure was raised to from 35 to 37 mm. of mercury. No choroidal pulsation appeared until the pressure was raised to from 48 to 50 mm. of mercury. This condition could be repeated as often as desired. The blood pressure of the patient was 135 systolic and 85 diastolic, and the electrocardiogram showed normal action of the heart.

From this Wegner concludes that the diastolic pressure in the choroidal blood vessels must be considerably higher than that in the retinal system.

F. H. ADLER.

HEAT CURRENTS IN THE ANTERIOR CHAMBER. S. KAWAHARA, Acta soc. ophth. jap. **36:** 29 (April) 1932.

In the normal eye the heat currents in the anterior chamber cannot be readily seen. Under pathologic conditions they are easily visible with

the slit lamp. The cellular elements that appear in the anterior chamber in the case of an acute or chronic iridocyclitis appear as fine, glistening points. In certain cases it is noted that these points do not stand still but slowly ascend along the posterior part of the anterior chamber, and then as they approach the posterior surface of the cornea they sink. This is caused by an associated movement of the aqueous which is related to the conditions of warmth within the anterior chamber. Kawahara concludes:

1. In the course of iridocyclitis, episcleritis, keratitis, herpes, foreign bodies or injuries, cellular elements may appear in the anterior chamber like fine, glistening, circulating or stationary points.

2. The rate of progress of the circulating dots varies between 0.5 mm. in a second and no movement.

3. The rapidity of the current depends on the concentration of albumin in the aqueous humor, on the temperature outside and on the depth of the anterior chamber. If the external temperature and the depth of the anterior chamber remain constant the rapidity of the current is slower in proportion to the amount of albumin. These dots are deposited on the posterior surface of the cornea as well as on the anterior surface of the lens, and are of a disklike structure in which blood cells and fine granular uveal pigment are grouped together. At the periphery the margin is less regular, and the deposits consist principally of uveal pigment.

4. The study of the circulation of these particles in the anterior chamber is of great clinical importance in intra-ocular diseases and is of assistance in estimating the course and prognosis. A. KNAPP.

Refraction and Accommodation

INFLUENCE OF AMETROPIA ON CERTAIN INFECTIONS OF THE ADNEXA OF THE EYE (LIDS, LACRIMAL TRACT). G. ROBERT, Ann. d'ocul. **170:** 633 (Aug.) 1933.

Prolonged fatigue of the eyes, even though slight or caused by ametropia of slight degree, in certain cases affects the lids and the lacrimal tract and the eye itself. In these cases there is asthenopia, varying according to the age of the patient, and infectious processes are also found, particularly if the surrounding cavities are infected, making the patient susceptible.

In the presence of infectious manifestations, it is necessary to make a thorough examination to determine the refraction and to correct this as nearly as possible. Slight degrees of ametropia should not be neglected. Except in rare cases a normal eye as a whole is not easily susceptible to infection; so if infection appears, and particularly if it becomes chronic, it is necessary to look for a local cause. One should begin by correcting the ametropia and follow this by the usual orthodox treatment.

These ideas are the same as those expressed by Lagrange on the necessity of complete refractive correction as the basis of all satisfactory treatment of diseases of the eye. S. H. MCKEE.

Retina and Optic Nerve

RETINA IN HEMOCHROMATOSIS. K. MADDOX, Brit. J. Ophth. **17:** 393 (July) 1933.

Hemochromatosis consists of derangement of liver and possibly pancreatic metabolism whereby excessive deposits of iron and copper are laid down in almost all organs and tissues, including the ectodermal structures. A clinical picture of diabetes mellitus is produced, accompanied by a slaty-blue discoloration of the skin—"diabète bronzé."

In four cases of this disease the examination of the fundus oculi showed retinal pigmentary changes of varying degree. The deposition of pigment was of a diffuse and uniform character, but was especially concentrated about some or all of the margin of the disk. The edge of the disk, therefore, appeared exceptionally sharply cut, owing to the color contrast. The intensity of darkness was maintained through a radius of 3 or 4 mm. toward the periphery and then commenced to fade and assume the normal hue of a "tigroid" fundus. The pigment was a deep slaty-blue through which the retina appeared as a kind of ocher background. These appearances vary directly in depth as the skin changes. None of the other components of diabetic retinitis was seen. It is surmised that the pigment is hemofuscin, identical with that of the cutis externa. An accompanying colored drawing depicts the fundus.

W. ZENTMAYER.

A CASE OF ANGIO-GLIOMATOSIS RETINAE WITH PATHOLOGIC REPORT. T. A. CARR and H. B. STOLLARD, Brit. J. Ophth. **17:** 525 (Sept.) 1933.

The patient was a boy aged 13 years. The left eye showed vitreous opacities and a circular white woolly-looking mass situated in the retina and encroaching on the upper temporal quadrant of the optic disk. Six months later the white mass was larger and was raised from 4 to 5 diopters, and vision was reduced to the ability to count fingers at 3 meters. One year later the mass was much larger and had an elevation of 7 diopters, and two small hemorrhages were noted. All tests were negative. A diagnosis of neoplasm was made, and the eye was enucleated. The pathologic report was as follows:

There was a neoplasm arising from the retina to the temporal side of the optic disk, and invading the papilla. It was shaped like a collar stud; the basal diameter was 2.5 mm., and the anteroposterior depth, 4.5 mm. It was composed mainly of endothelial cells, arranged in irregular clumps, and of glial tissue. Near the summit of the neoplasm there were several cystic spaces filled with granular débris, fibrin, lymphocytes and large mononuclear leukocytes. The walls of these spaces were formed by endothelial cells and glial tissue. Several small capillaries could be seen passing into these areas of cystic degeneration. At the base of the neoplasm there were many amyloid bodies scattered irregularly, and at the temporal edge of the optic disk there was a mass of bone arising from the choroid and passing forward into the base of the neoplasm.

The contiguous retina was rucked, and there was a small collection of subretinal fluid which produced a shallow localized detachment. Some punctate deposits of leukocytes were present on the back of the cornea, and the anterior surface of the iris and the ligamentum pectinatum showed a plasma cell and lymphocytic infiltration. The remainder of the eye was normal.

There was no evidence of extra-ocular extension of the neoplasm. The authors believe that it was relatively benign.

A diagnosis of angiogliomatosis with degenerative changes was made. The points of interest are that no other lesion was found elsewhere in the body on clinical examination, and that the neoplasm grew slowly; the histologic picture suggested that it possessed a relatively benign character and was even retrogressive in the parts where cystic degeneration and formation of bone and amyloid bodies had taken place.

W. ZENTMAYER.

REGENERATION OF THE RETINA AFTER ITS OPERATIVE REMOVAL. M. BÜCKLERS, Arch. f. Ophth. **130: 257** (Aug.) 1933.

The author removed the entire retina or parts of it from the eyes of Rana temporaria during the larva stage, and studied the process of regeneration. After partial extirpation, the defect was at first filled in by proliferating pigment epithelium which also formed one continuous layer toward the vitreous. At the same time the ganglion cells of the retina surrounding the defects began to proliferate and formed a multilayered stratum of cylindric, very immature cells. When the stratum had reached the thickness of the normal retina, the process of differentiation set in. The regeneration produced an excess of retinal tissue which protruded into the interior of the eye. After total extirpation the regeneration extended from two points of the posterior surface of the iris, one located dorsally, the other ventrally. The process of regeneration was not disturbed or influenced in any way by the metamorphosis. "Rosettes" were nearly always found in the regenerated portions. The vitreous, which was unavoidably destroyed in these experiments, was reformed by the cells of the hyaloid membrane and by the blind portion of the retina.

P. C. KRONFELD.

SO-CALLED ATROPHIC DECOLORATION OF THE DISK. L. SCHREIBER, Arch. f. Ophth. **130: 312** (Aug.) 1933.

Knowledge of the various anatomic processes which may produce the ophthalmoscopic picture of a pale disk is still incomplete. Atrophy of the nerve fibers is by no means the only factor which can make the disk appear gray or white. Proliferation of the glial and mesodermal supporting structures and anemia (capillary spasm) have been mentioned as possible causes. Schreiber suggests a new possibility, namely, an acute swelling of the nerve fibers. He bases his suggestion on a number of significant clinical and pathologic observations.

1. Some types of white decoloration are reversible. This was seen in cases of multiple sclerosis by Behr. Schreiber examined a girl with

dystrophia adiposogenitalis whose disks were found to be "atrophic" white on two occasions, and each time they returned to normal after roentgen treatment of the hypophyseal tumor.

2. Five days after fractures of the skull or traumas of the orbital portion of the optic nerve the disk may appear white, although a Weigert stain at that time shows hardly any visible atrophy.

3. Jaburek, in studying the pathologic changes in multiple sclerosis, found swelling of the axis-cylinders to be the first sign of the pathologic process. This swelling, if it did not extend beyond a certain limit, was reversible.

4. Schreiber, in a previous paper (*Ztschr. f. d. ges. Neurol. u. Psychiat.* **4:** 386, 1911), demonstrated reversible changes of the optic nerve fibers which he named the Marchi reaction. The myelin sheaths stained with osmic acid but showed no breaks in their continuity, which is characteristic of the Marchi degeneration. An eye affected with a serpiginous ulcer of nine days' duration showed chromatolysis in the ganglion cells of the retina and a pronounced Marchi reaction in the optic nerve. Schreiber assumes that these changes would have been reversible because the vision in eyes which have recovered from serpiginous ulcers is usually determined only by the extent of the corneal scar.

In the practice of ophthalmology it seems highly advisable to differentiate between so-called atrophic decoloration of the disk, which is reversible, and the irreversible, really atrophic pallor.

P. C. KRONFELD.

RETINITIS PIGMENTOSA AND MULTIPLE SCLEROSIS: REPORT OF A CASE. F. RIECHERT, Klin. Monatsbl. f. Augenh. **91:** 163 (Aug.) 1933.

A young man, who was healthy up to his sixteenth year, began to lose his vision. There was no anamnesis of heredity, and he did not have syphilis. Vision decreased more rapidly from his twentieth year on; uncertain gait developed four years later, followed by other symptoms of multiple sclerosis.

Examination revealed retinitis pigmentosa. The disk, instead of presenting the yellowish tinge common to this disease, was pale, resembling the appearance in total optic atrophy. Bilateral central scotoma for white and colors and concentric contraction of the visual fields with peripheral gyric remnants of vision were noted.

The progress of the retinitis and the sclerosis was simultaneous. Judging by the unusually grave lesion of the optic nerve, Riechert suspects that both diseases were caused by hereditary inferiority of the tissues.

K. L. STOLL.

MACULOCEREBRAL TYPE OF HEREDODEGENERATION OF THE MACULA LUTEA. R. BRAUN, Klin. Monatsbl. f. Augenh. **91:** 182 (Aug.) 1933.

After excluding the Tay-Sachs and Vogt-Spielmeyer-Stock diseases, and also syphilitic chorioretinitis and retinitis pigmentosa, the author finds that genuine degeneration of the macula lutea may or may not

be accompanied by symptoms of involvement of the central nervous system. These symptoms may be so slight in some cases that only an exact neurologic examination may reveal them. He reports a case of this description. A healthy young woman, aged 23, had had faulty vision since childhood. Vision decreased for about nine months prior to admission to the hospital, when it was 1/8 of the normal in the right eye and 1/10 in the left. Central scotomas for colors were present in both eyes and slight eccentric fixation. Bilateral round foci of the size of the disk were seen in the macular region; yellowish dots were noticed within these foci and pigment in their surroundings. The temporal portion of the disks showed slight pallor. Neurologic examination revealed slight weakness of the right facial nerve, left-handedness and facility in mirror writing with her left hand, with which she wrote regular script when a child. No disturbance of speech was noticed when she began to write with her right hand. Her mother and a sister suffered from occasional epileptic attacks. Braun considers this a borderline case of the type described by Behr.

K. L. STOLL.

Tumors

SARCOMA OF THE CHOROID. E. ANNEN, Ann. d'ocul. **170:** 651 (Aug.) 1933.

Sarcomas of the choroid may be classified macroscopically into three groups: pure melanosarcomas, pure leukosarcomas, pure and mixed forms. The mixed forms are by far the most frequent.

Pure melanosarcoma has had the reputation of showing the greatest malignancy. Nevertheless, perforation and metastases have been found in connection with other types of sarcoma in as many, if not more, cases. In other words, malignancy is not related to pigmentation.

A tumor of the choroid does not as a rule invade the sclera but grows along the choroidal vessels and chooses the line of least resistance in the globe.

The degree of detachment of the retina bears no relation to the size of the tumor; a small sarcoma may cause almost total detachment, and vice versa.

Spontaneous detachment of the retina on examination with the ophthalmoscope may give the appearance of a tumor, particularly when cysts or posthemorrhagic deposits are present.

S. H. MCKEE.

THE SYNDROME OF TUMORS OF THE APEX OF THE ORBIT. JEAN SÉDAN, Ann. d'ocul. **170:** 846, 1933.

The syndrome of the sphenoid fissure was first described in detail by Rochon-Duvigneaud in 1896. This contribution was followed by the complete study of the subject by Professor Rollet and his pupils, Berthoux and Debergue, and Professor Terrien and his pupil, Casteran. With involvement of the optic canal, Rollet proposed the term "syndrome of the orbital apex."

The syndromes of the sphenoid fissure and orbital apex have a sensory symptomatology which is most important in reaching a diagnosis. Casteran in his thesis says that it is the sensory disturbances that give the name to the syndrome. These disturbances are well known and classic, and there has been nothing added in numerous published descriptions of the anesthesias in the trigeminal territory in the course of a lesion at the apex of the orbit. The anesthesia is generally evident, but often the patient directs one's attention to this point.

Prof. Roger de Marseille seems to have been the first to insist on the importance of headache in this syndrome. Having had eight cases to examine, he was struck with the fact that headache was constant in them all. Even after correction of errors of refraction, diplopia and even ptosis, there still remained unpleasant and persistent headache, which was the first symptom observed. Complete corneal anesthesia is rare, but diminished sensation is the rule.

S. H. McKee.

Uvea

"Atrophia Gyrata Choroideae et Retinae" in High-Grade Myopia. A. Ohashi, Acta soc. ophth. jap. **36:** 53 (June) 1932.

The condition observed by Ohashi differed from that described by Fuchs in that it was stationary and without familial history, was unaccompanied by other congenital anomalies, and gave rise to a peculiar form of night blindness.

A. Knapp.

Visual Tracts and Fields

Minute, Permanent Annular Scotomas After Traffic Accidents, and Paracentral Scotoma After Electric Examination of the Ear: Report of Cases. A. Fuchs, Klin. Monatsbl. f. Augenh. **91:** 20 (July) 1933.

Two cases are reported and compared with an analogous case of Wisselink's, in which contusion of the face was followed by minute annular scotomas in one eye. One man, aged 30, noticed a blur in one eye two days after an airplane accident. A small central scotoma of annular shape was found about a year later. The second man, aged 25, noticed a central, ring-shaped defect immediately after an automobile accident. Neither patient was unconscious after the accident, nor was their vision impaired. Lesions of the eyes, the disks and the skull and contusion of the brain were absent. Faint changes in the fovea could be detected with red-free light only in one case. Wisselink's case was similar to Fuchs' first case, while a related case of Betsch is cited by the author as a more serious condition of the same type.

After ruling out hemorrhages in the sheath of the optic nerve or in this nerve directly, Fuchs thinks that the disturbances were caused by minute tears in Henle's fibers with consecutive edema and possibly scant admixture of blood.

A man, aged 50, experienced the apparition of a strong light and a shock while an otologic test of his labyrinth was made with a weak

electric current. No pain was felt, but the patient noticed a blur when reading a few days later. A small central scotoma was found, but no other pathologic changes. Stefan Jellinek, who was consulted as an expert in physics, referred to lesions caused by an electric current passing through the skull. He warned against the use of the street current in these otologic tests, which should be done with currents from batteries only.

K. L. STOLL.

Therapeutics

MANAGEMENT OF TABETIC OPTIC ATROPHY. G. F. SUKER and M. M. JACOBSON, Illinois M. J. **63**: 21 (Jan.) 1933.

Suker and Jacobson state that according to some statistics tabetic optic atrophy has not been increasing. More careful observation of the results obtained in the management of general systemic syphilis is indicated, so that proper changes in management can be made in order to prevent some of the late developments in the central nervous system. Intracranial injections of mercuric chloride have given better results than any of the other methods used. There have been several cases in which definite improvement or arrest of the condition has followed with the retention of useful vision for a period of several years. A negative Wassermann reaction of the spinal fluid has resulted frequently. The method of choice is the intraventricular injection which, in a large measure, is to be considered as a topical application and is a relatively safe procedure. In order to accomplish the utmost benefit in the management of tabetic optic atrophy, the diagnosis must be made early; every patient with syphilis of any number of years' standing should be critically examined with reference to tabes and dementia paralytica; in every such case, careful perimetric examination should be made to determine the earliest possible infringements of the visual fields, and the immediate application of intracranial injections should be followed by systemic treatment as well.

[J. A. M. A.]

DIATHERMY IN DISEASES OF THE EYE. L. SHERESHEWSKAYA, Sovet. vestnik oftal. **1**: 14, 1933.

The author treated twenty-three patients with diathermy, using the Bucky electrode, with the strength of the current from 0.2 to 0.4 amperes. Each treatment lasted from five to ten minutes, and the number of treatments varied from ten to thirty.

Five patients with blepharoconjunctivitis and two with dendritic keratitis did not respond to the treatment. The most favorable results were obtained by the author in four cases of episcleritis and ten cases of iridocyclitis. All the acute symptoms disappeared after five or six treatments. In one patient suffering from tuberculous choroiditis in whom specific therapy gave no evident results, the lesion of the fundus partially absorbed and the vision improved from 0.02 to 0.3 after twelve diathermy treatments.

O. SITCHEVSKA.

Toxic Amblyopia

Toxic Amblyopia and Accompanying Physiological Disturbances in Carbon Tetrachloride Intoxication. Zolton T. Wirtschafter, Am. J. Pub. Health **23:** 1035 (Oct.) 1933.

Five cases of poisoning with carbon tetrachloride are reported. In all of them there was a concentric contraction of all the color fields without any central scotomas. Unfortunately no records of the visual acuity are included in the report. When the patients were removed from exposure to carbon tetrachloride and placed on a diet high in calcium and dextrose the amblyopia rapidly disappeared.

Francis H. Adler.

Society Transactions

EDITED BY DR. JOHN HERBERT WAITE

NEW YORK ACADEMY OF MEDICINE
SECTION OF OPHTHALMOLOGY

Jan. 15, 1934

WEBB W. WEEKS, M.D., *Chairman*

GUERNSEY FREY JR., M.D., *Secretary*

VERNAL CATARRH. DR. ISADORE GOLDSTEIN.

B. G., a woman, aged 22, has had vernal catarrh in both eyes for the past fourteen years. During this period she has been under constant treatment, including the usual local applications, radium irradiation and diathermy, without improvement. In April 1933 the entire tarsus and the diseased portion of the conjunctiva were removed from the left upper lid and, in their place, mucous membrane taken from the lower lip was transplanted. Following the transplantation there was a gradual disappearance of the itching and discomfort with a lessening of the discharge. The thickened conjunctiva of the lower lid has become normal. Up to the present time, no recurrence has taken place. Three months ago a similar procedure was performed on the right eye, so that, at present, the patient is free from symptoms.

DISCUSSION

DR. DAVID H. WEBSTER: My associates and I had four cases in which we excised the tarsus and conjunctiva, but we have not at the Manhattan Eye, Ear and Throat Hospital implanted mucous membrane from the lower lip.

DR. JOHN M. WHEELER: Physicians have been so nearly helpless in treating vernal catarrh that it seems to me one ought to welcome anything that has promise. This patient of Dr. Goldstein's has apparently obtained relief after a history of many years. Dr. Goldstein said that such a radical excision of the tarsus may lead to sloughing of the lid margins. I have seen sloughing of the lid margin from too thorough excision. I wonder if it is feasible to do the operation in two stages, first cutting away the upper part of the tarsus and making a transplant, and then later cutting away the lower tarsus and making another transplant.

DR. GOLDSTEIN: In the left eye I did the upper part first, but from the lower remaining portion there was an extension of the lesion into the new graft. Since this occurred, I have decided it is best to remove the entire tarsus and as much of the conjunctiva as is necessary.

DR. WHEELER: How much contraction did you allow for?

DR. GOLDSTEIN: I did not allow for any contraction. The graft was about 30 mm. long and 20 mm. wide.

DR. MARTIN COHEN: Several years ago, Dr. Wootton and I showed at a meeting of this section a patient with vernal catarrh in whom extirpation of the conjunctiva and tarsus had been performed. The classic symptoms of vernal catarrh recurred a year later.

EXCISION OF STAPHYLOMA OF THE CORNEA. DR. DANIEL B. KIRBY.

A protruding staphyloma of the cornea developed after perforation of a corneal ulcer in a young girl. After freeing the anterior synechia, iridectomy was performed. Seven months later, an elliptic piece of cornea was removed by a combination knife and scissors section. Apposition of the edges was obtained by sutures, and a conjunctival flap was used. Healing was by primary union with a good result in the shaping of the cornea, preparing it for the reception of a corneal transplant.

DISCUSSION

DR. RAMON CASTROVIEJO: Dr. Kirby has proved that in these cases in which enucleation is generally advised everything should be done before removing the eye. With present knowledge, certain eye conditions cannot be cured, but a cure for them may be found in the near future. An eye which has perception and projection of light should not be enucleated unless it is painful or dangerous to the other eye or to the life of the patient.

TRANSPLANTATION OF CORNEA WITH PRESENTATION OF A CASE AND A MOVING PICTURE OF THE OPERATIVE TECHNIC. DR. RAMON CASTROVIEJO.

The speaker outlined a new technic which he had employed for transplantation of the cornea and reviewed the results obtained in seven patients. Preliminary to operation, blood grouping was done in donor and host. One eye was lost from postoperative infection. The transplant took well in the remaining six patients, but Dr. Castroviejo did not wish to commit himself until a larger series of transplants had been observed.

DISCUSSION

DR. BEN WITT KEY: It is my conviction that transplantation of the cornea is dependent not only on delicate surgical skill, but also on a background of chemical knowledge. For many years it has been regarded as a surgical problem dependent on the nourishment of the transplant. With this in mind, it was my purpose to supply nourishment by transplanting the whole cornea together with a large circumcorneal conjunctival flap. Although the corneoconjunctival transplant took hold completely, the cornea very gradually became opaque over a period of two years. The conclusion to be drawn from this is that the transplanted Descemet's membrane becomes altered and permits the entrance of aqueous into the corneal substance. If I had another patient with complete corneal opacity I should try to dissect the cornea of the donor and the cornea of the host, stripping the stroma from Descemet's membrane

in each case. If one could possibly prevent extensive contact of the transplanted corneal substance with aqueous, I believe one might succeed in having transparency maintained.

DR. ISADORE GOLDSTEIN: I saw Sir Tudor Thomas' work at Cardiff, England, two years ago, and at that time he was getting satisfactory results in rabbits. Not alone was he transplanting the cornea, but also the sclera. Recently he has done some work on man with very good results.

DR. RAMON CASTROVIEJO: In these experiments I have found that the transplants which have remained transparent have normal histologic structures, including the epithelium. Asher, in 1922, proved that the trephine produces irregular corneal incisions. I arrived at the same conclusion, and also concluded that linear incisions are cleaner than the ones made with a trephine.

I feel, as Dr. Key does, that the aqueous humor is the enemy of the cornea. If the size and shape of the transplant are perfect and it fits the defect of the cornea of the host, it will make almost a water-tight closure. The endothelium of the transplant and that of the cornea of the recipient will be in direct contact and will regenerate rapidly, covering the gap made by the incision and separating the aqueous humor from the stroma. The endothelium seems to be more important than Descemet's membrane for the protection of the corneal stroma.

The method suggested by Dr. Key, of peeling the corneal scar down to Descemet's membrane and endothelium and placing in this bed the superficial layers of the whole surface of the cornea with a rim of conjunctiva obtained from the donor's eye, seems almost impracticable in the light of present knowledge. Connective tissue has to develop between the cornea of the recipient and that of the donor with subsequent loss of transparency of the transplant.

RETINOBLASTOMA. DR. M. JAFFE.

This article will be published in full in a later issue of the ARCHIVES.

METASTATIC CARCINOMA OF THE RETINA. DR. JULES W. SMOLEROFF.

This article will be published in full in a later issue of the ARCHIVES.

Feb. 19, 1934

WEBB W. WEEKS, M.D., *Chairman*

W. GUERNSEY FREY JR., M.D., *Secretary*

CHOROIDAL ATROPHY WITHOUT NIGHT BLINDNESS. DR. ISAAC HARTSHORNE.

A 7 year old girl complained of poor vision of one year's duration, apparently not progressing and without night blindness. There had been three attacks of severe prostration in 1929, 1930 and 1931, with

drowsiness followed by semicoma and acetone breath. Following the third attack she had foul, fatty stools and persistent jaundice. An attempted Wassermann test on the patient was unsuccessful; the father's Wassermann reaction was negative, and the familial history was negative for syphilis.

The physical examination and blood picture showed normal conditions. Corrected vision was 20/50 in each eye. The media were clear. The ophthalmoscope revealed: diffuse peripheral choroidal atrophy, with the larger choroidal vessels very prominent against the white scleral background; many pinpoint-sized and larger flecks of pigment migrated from the pigmented epithelial layer to inner layers of each retina, especially peripherally and around each macula; the retinal vessels not remarkable; the disks normal, and no hemorrhages or exudate. The visual fields were full.

This case confirms Dr. Verhoeff's statement that changes in the choroid do not produce retinitis pigmentosa. In this case, lacking night blindness, the rods were evidently intact in spite of choroid degeneration. The pigment had migrated into layers of the retinae but had not followed a blood vessel outline. The changes in the pigmented epithelium were probably due to whatever caused the choroidal atrophy, rather than to degeneration of the neuro-epithelium which, in this case, was at a minimum.

Considerable evidence pointed toward hepatic malfunction as a toxic cause of this condition, the toxic effect possibly arising from an increase in the blood of bile salts or other substances derived from the liver.

COATS' DISEASE. DR. R. TOWNLEY PATON.

H. B., a white boy, aged 6, was first seen on Nov. 7, 1933. The familial history was noncontributory, and there was no history of tuberculosis or of rheumatic fever. Some time during the summer of 1933 the parents noticed that the vision in the left eye was markedly diminished. During the early part of September a yellowish mass first appeared in the left eye. There was no pain or photophobia, and no physical symptoms were noted.

External examination at the first visit revealed the right eye to be normal.

The left eye lacked light perception, and the left pupil was slightly dilated, not reacting to light or during accommodation. The left tension was normal with the Schiötz tonometer. Focal illumination of the left vitreous gave a grayish reflex, due to a nonvascular mass projecting forward, more peripherally than at the center. Transillumination showed a shadow, more marked below on the nasal side, though the transillumination was poor in the entire circumference. Practically the whole left fundus was occupied by a billowy mass of exudate, with complete detachment of the retina. The left optic disk, though indistinctly seen, appeared normal in size, shape and color. Only a few retinal blood vessels could be made out. In the central portion of the fundus, almost in line with the optic disk, could be seen a retinal vessel, which appeared to terminate in an aneurysmal dilatation. Irregularities of caliber, but no marked dilatations, were noticed in the other vessels. There were present a number of glittering spots, suggestive of cholesterol

deposits. The most anterior portion of the detached retina could best be seen with a plus 10 sphere.

In the differential diagnosis, the following lesions were considered: glioma, tuberculoma, metastatic abscess from infected tonsils and angiomatosis retinae. The physical examination showed no contributing causes for the condition of the eye; x-ray pictures of the chest and sinuses were normal; a tuberculin test was negative, and the Wassermann and the Kahn test were negative.

DISCUSSION

DR. EMANUEL M. JOSEPHSON: I wish to suggest a means of differential diagnosis between Coats' disease and detachment of the retina due to other causes. In the regions of maximum involvement in Coats' disease the exudate forms about the vessels and overlies them. The finding of engorged veins in the regions of the retina not directly involved in the exudative process, due to the blocking of the vessels in the involved area, is a well recognized pathologic feature of the disease. In contrast, normal-appearing vessels are regularly found coursing over the surface of a simple retinal detachment.

DR. ARTHUR J. BEDELL: It seems to me that Coats' disease is not an entity. I think the members will agree that from Dr. Paton's pictures the diagnosis should be, using the most recent terminology, angiomatosis retinae, for the angioma was visible on the slide. It must be remembered that since Coats wrote his masterly article many new clinical and pathologic facts have been collected.

HEREDITARY AND FAMILIAL DEGENERATION OF THE MACULA. DR. RALPH I. LLOYD.

Many macular changes occurring throughout life are now regarded as degenerative processes. When the female is the transmitter she does not escape as in some other inherited ailments of the eye. The time of the appearance of the lesion is always the same for the members of the one family. Most of the cases are juvenile, but there are many other periods in which cases occur. Some cases begin at birth like those of the Tay-Sachs type, others at dentition and others at puberty, and then follows the period of sex maturation and the end of skeletal growth. The senile cases, originally considered as a separate entity, are now, like the presenile cases, thought to belong to the group of complaints obedient to hereditary influences. The milder group includes the choroiditis guttata of Tay and the honeycomb choroiditis of Doyne. The more serious types include the maculocerebral degeneration (familial) of Oatman, Tay-Sachs' disease, the familial congenital macular degeneration of Best, the progressive macular degeneration of Batten and Stargardt and the tapetoretinal degeneration described by Leber.

After middle age, there seems to be commonly a change in the elastic tissue of the body. Senile patients have regularly a lessened efficiency of the finer arterioles due in large part to changes in the elastic layer. The well known drusen found in the eyes of elderly people represent a very marked change in the elastic layer of the choroid, and the same hyaline layer found in "guttate choroiditis"

beneath the pigment layer of the retina is found in senile changes. It is now believed that eventually these benign changes found in *Drüsen* and also in the mild type of hereditary macular degeneration may have an effect on the vision as a result of the continued interference with the nourishment of the rods and cones. How early these hereditary macular degenerations appear cannot be said, but the cases shown here tonight were observed in persons ranging in age from 15 years to 50.

In striking contrast with these apparently benign cases are the less frequent cases with serious changes in the vision and sometimes associated with progressive mental deterioration. When first recognized, these cases are characterized by a "pepper and salt" macula. Later, the normal pigment layer of the retina and the superficial layers of the choroid begin to thin in the region about the disk. The vision fades well in advance of the macular changes, and a ring scotoma develops with the very small functioning area at the macula showing defect for blue first, while still able to recognize red. If these cases begin before puberty, they pass as a rule into an imbecile state. If the lesions begin after puberty, the mental deterioration is usually absent.

Macular colobomas like other colobomas of the choroid may be familial. The tendency in Europe is to look on the macula changes of senility as familial. It is certain that senile macular degeneration is but one of a group of symptoms resulting from vessel changes in the brain.

Considered as a separate entity and at first as anything but a familial disease, the condition of "angioid stripes in the retina" presents a presenile macular disease which must be considered in connection with the previous group. It rarely begins before the fourth decade and like the other group is familial. The progress is very slow, running for from five to twenty years. The feature of this disease is the stripe or streak or group of such surrounding the macula with branches running outward in all directions. The color of the streaks is red, brown or even almost white. The vision is affected early, and the progress is steady to loss of central vision, by which time the second eye is involved and on its way to blindness. Several members of the family are affected, but the fundus picture of the family group is not uniform.

DISCUSSION

DR. HENRY H. TYSON (after discussing the early recognition of "angioid streaks" by Plange and Herman Knapp): The pathologic process is probably a metamorphosis of deep choroidal hemorrhages. In retinal pigment degeneration at the macula, the fundus changes are not unlike those that occur in the aged, as though the retina were affected with a local premature senility. The absence of all evidence of inflammatory action suggests that the retinal atrophy results from nutritive disturbances. Inasmuch as the retinal layer first affected is nourished entirely by the choroid it might be inferred that the primary lesion is in the choriocapillaris. Furthermore, limitation of the pathologic changes to the posterior pole of the eye corresponds to the distribution of a choroidal vascular circuit.

DR. CONRAD BERENS: I shall limit my discussion to the probable etiology and pathology of certain lesions of the macula.

Lantern slides of several senile macular lesions which I present illustrate that although hemorrhages which have been recurrent are often possibly prevented by the elimination of foci of infection, the majority of these patients had progressive loss of vision, generally to 20/200. The reason for this is, I think, that only certain of the infections can be completely eliminated. The bacteriologic studies which I have demonstrated indicate that certain patients whose tonsils and teeth had been removed and who had received thorough intestinal and general treatment still harbored great numbers of pathogenic streptococci in the nasopharynx and intestines.

I think that angioid streaks in the retina are best explained by the observation of Collins that they are the results of hemorrhages in the choroid. Verhoeff's pathologic section, which I show, possibly demonstrates that the angioid streaks are caused by ridges in the inner layers of the choroid produced by cicatricial contraction of fibrous tissue which had replaced the deeper layers.

DR. ERNST WALDSTEIN: Two cases demonstrated tonight were those of a young man and his sister, both in the early twenties; one had a macular degeneration; the other, an aplasia of the macular region with lack of a macular and foveolar reflex and the presence of nystagmus. None of these patients with macular degeneration whom we had the privilege of seeing tonight had nystagmus. These cases prove that two absolutely different kinds of macular affection (aplasia and degeneration) may originate in the same family.

W. GUERNSEY FREY JR., M.D., *Secretary*

March 19, 1934

WEBB W. WEEKS, M.D., *Chairman*

DIVERGENCE EXCESS. PROF. ALFRED BIELSCHOWSKY (by invitation).

This paper appears in full, with the discussion, in this issue of the ARCHIVES.

ROYAL SOCIETY OF MEDICINE, LONDON, SECTIONS OF OPHTHALMOLOGY AND DERMATOLOGY

Joint Meeting, Feb. 9, 1934

MR. A. C. HUDSON, F.R.C.S., *Chairman*

CONDITIONS COMMON TO OPHTHALMIC AND DERMATOLOGIC PRACTICE

OPHTHALMOLOGY. MR. J. H. DOGGART.

Numerous diseases in the field of general medicine commonly involve the skin and the eyes. It is well known that in measles there may be conjunctivitis and possibly corneal ulcers, in addition to the rash.

Hypofunction of the thyroid gland has been known to produce multiple opacities in the corneal substance, associated with the characteristic myxedematous changes in the skin, and both of these manifestations have simultaneously disappeared after a course of treatment with glandular extract.

Blepharitis may be of any grade of severity. At onset, it is usually due to some variety of conjunctivitis or of facial dermatitis, but if it is neglected or improperly treated, blepharitis can persist by itself. The longer the condition endures, the more it approaches the ulcerative form, which is characterized by scantiness, irregular growth and misdirection of the eyelashes. When blepharitis is persistent it is advisable to test refraction and to correct errors, though the etiologic importance of refractive errors has been exaggerated. In this way, the patient is prompted not to rub his eyes and so cause a continuance of the irritation. In many cases blepharitis is aggravated by overtreatment. All strong solutions must be avoided. Thrice daily one may use lavage with physiologic solution of sodium chloride, followed by application of petrolatum or ointment of weak boric acid to the lid margins.

By the layman acne rosacea is erroneously linked with alcoholic indulgence. The keratitis of acne rosacea is an obstinate disease, with a tendency toward relapse. There is occasional perforation of the cornea. Most patients with the condition come to have at some time a mild blepharitis not associated with distortion of the lid margins, but predisposing to the formation of meibomian cysts. The severity of the attacks may diminish with age. The value of treatment is difficult to estimate. An apparent cure following the use of a particular remedy may mean simply that the case has been caught at a period when the condition was naturally involuting. No method should be recommended until it is proved to have been of value during acute relapses in a number of consecutive cases.

DERMATOLOGY. DR. H. C. SEMON.

In relation to acne rosacea so many things have been alleged to be predisposing factors that the only way to reconcile them is to predicate nervous disturbances at the periphery of a kind of reflex arc in the sympathetic nervous system. The flushings suggest an intervention or an association of angioneurotic factors. The abnormal stimuli ultimately lead to chronic stasis and congestion, which the capillaroscope shows. The congestive state leads to disturbances of secretion and predisposes to infection of the sebaceous glands, at which point the symptoms of acne rosacea become apparent. This conception is further supported by the fact that the symptoms are aggravated by emotion, cold, heat and light. A seborrheic subject is especially liable to have acne rosacea. Acne rosacea is relieved in most cases by dietetic means. The patients must rigidly abstain from alcohol, coffee, strong tea, condiments and pickles, and drinks are best taken cold or tepid. Constipation and all septic foci must be eliminated. Local applications of a calamine lotion containing sulphonated bitumen and a lead compound (1 per cent) are valuable in treating an acute or irritable condition. For the more chronic varieties, sulphur and resorcinol are good. A resistant condition or one in which a seborrheic element is pronounced is usually benefited by carefully graded doses of x-rays.

Dermatitis of the eyelids may be primary or secondary. The eyelids are very sensitive, and frequently are the first to show signs of irritation by exogenous irritants, such as hair dye, plants, powders, scents or the vapors of formic or osmic acid, to which the patient may be peculiarly sensitive. Once acquired, the allergy of the patient may be extreme. Infections of the scalp and of the face are often associated with marginal blepharitis, and the degree of eyelash destruction in any case of chronic seborrheic dermatitis is an index of the severity of the infection, and hence a guide to the prognosis. Treatment must be persistent, for in few cases is a well established, chronic acne rosacea really cured.

DISCUSSION

MR. R. AFFLECK GREEVES: The ocular lesions of acne rosacea resemble those of phlyctenular disease. Peripheral corneal lesions are common, and the ulcers of the cornea are very resistant to treatment. Lesions like those in phlyctenular disease respond best to stimulation by an ointment containing yellow oxide of mercury, with administration of atropine to relieve the pain of photophobia. I object to phenolizing the ulcers. The greatest relief is afforded by irradiation with the x-rays, in doses of from one sixth to one third of a Sabouraud pastille, not more often than once a fortnight. Early irradiation with the rays may cause a recent infiltrate to disappear, and there is no reaction to such a small dose.

DR. SYBIL EASTWOOD: In 95 per cent of the cases of acne rosacea examined in the department for skin diseases at University College Hospital there was some digestive disturbance. In long-standing acne rosacea, the underlying factor was found to be a vascular congestion, in most cases arising from the stomach. Forty-eight per cent of the patients had achlorhydria. The most promising cases were those in which the secretion of hydrochloric acid was low, for administration of hydrochloric acid made the patients well and kept them so or, at least, with only mild recurrences. The gastric condition should be investigated in every case.

DR. S. H. BROWNING: Many persons with seborrhea come to me for bacteriologic examination, and a large proportion have seborrhea of the scalp or pityriasis capitis, the treatment of which produced good results. Most of the blepharitis which I see is staphylococcic, usually being due to Staph. aureus, sometimes to Staph. albus. Vaccines, I find, are an adjunct to other forms of treatment. Styes may be unassociated with any other staphylococcic condition, and ordinary treatment usually suffices.

KELOID. DR. A. J. DURDEN SMITH.

The value of treating keloid with radium has been shown in five cases. No second exposure should be given until the full effect of the first has had time to develop. In keloid in the orbital region, implanting radon seeds avoids the danger of postirradiation changes in the eye.

DISCUSSION

MR. A. C. HUDSON: One sometimes sees burns and keloid formation between the side of the nose and the inner part of the lower lid. Irradiations with radium will often soften keloid.

Unusual Coloration of the Sclerotic Coat. Mr. P. G. Doyne.

An actress noticed that her eyes had gradually been turning blue. The slit lamp showed a fine punctate deposit on the bulbar conjunctiva. The patient did not use a silver stick for make-up, but her grease paint had silver in it, and the condition may be argyrosis.

ROYAL SOCIETY OF MEDICINE, LONDON, SECTION OF OPHTHALMOLOGY

Clinical Meeting, March 9, 1934

Mr. A. C. Hudson, F.R.C.S., *President*

Osteoma of the Ethmoid. Mr. Duncan Fitzwilliams.

The patient, 51 years of age, complained of giddiness and of pain on the left side of the head. The left eyeball protruded for six months. There was difficulty in lowering the left upper lid. Roentgenograms revealed evidence of tumor in the ethmoid region. The pupils were equal in size, and the intra-ocular pressure was normal. There was some contraction of the left visual field.

A surgical approach was made by an incision from the center of the supra-orbital ridge to just above the inner canthus, dividing everything down to the bone. The bone was chiseled off the superior oblique pulley, leaving the pulley intact. The periosteum was elevated from the inner side of the orbit until the osteoma could be felt; then the osteoma was loosened from the thin bone at the inner side of the orbit. The wound was sutured, a small drain was placed deeply in the orbit, and a firm bandage applied. Double vision persisted for a month after the operation.

When the growth is a large one, especially one which encroaches on the cranial cavity, the transfrontal route is the only correct method of approach.

Diplopia from Paresis of the Left External Rectus. Mr. L. H. Savin.

A male attendant at a hospital for mental diseases received a severe blow in the left eye from a patient, and blood poured from a cut in the eyebrow. Diplopia was immediate and persistent. Paresis of the left external rectus was found with diplopia when he looked ahead or to the left. The fundi showed signs of old and diffuse choroidoretinitis. Neurologic examination disclosed nothing of significance. The Wassermann reactions of the blood and cerebrospinal fluid were negative. It seemed likely that the nerve lesion was traumatic. A full advancement of the left external rectus was carried out, with resection of the terminal centimeter of tendon. Shortly afterward diplopia could no longer be elicited.

TONIC PUPIL. MR. A. LISTER.

This condition is known under many names, and there are two forms: complete and incomplete. The only symptom complained of may be discomfort and, if the accommodation is lost, mistiness of vision. In most of the cases no hereditary or constitutional disorder is present. The cause is unknown. The onset may be either sudden or gradual. There are no complications, and improvement tends to take place in the course of years. In the cases in which complete paralysis of the pupil is present, a tonic contraction of the other may indicate the nature of the trouble. The Wassermann test should be taken for record. In all the cases the pupils are dilated. Neither diphtheritic nor Argyll Robertson paralysis bears any relation to this type of pupil.

INFANTILE CEREBROMACULAR FAMILIAL DEGENERATION (TAY-SACHS' DISEASE). MR. VICTOR B. PURVIS.

Four months ago a girl, aged 11 months, was taken to a children's hospital, because she was slow in picking things up. Both fundi showed the typical picture of Tay-Sachs' disease. The maculae stood out as dull red spots of one-third disk diameter, surrounded by a white opaque area extending to midway between the maculae and the disk. The optic disks were not yet grossly affected, but displayed definite pallor. She is the third and youngest child of Jewish parents; the parents are not consanguineous. The eldest sister died of Tay-Sachs' disease, but all other members of a prolific family have been free from the disease for three generations.

IRRADIATION AND CURE OF EPITHELIOMA OF THE LID BY RADIUM, WITH PRODUCTION OF LATE RADIUM CATARACT. MR. VICTOR B. PURVIS.

This patient, aged 52, complained of a swelling on the left upper lid which had been present for about two years. The tumor, 2.5 by 1.5 cm., infiltrated the left upper lid, had a broad base, and was raised about 1 cm. above the level of the normal lid. A small piece was excised for biopsy. One hundred and twenty hours of continuous radium therapy was applied, screened to produce pure gamma irradiation, to insure adequate and uniform penetration and at the same time to avoid necrosis. The tumor disappeared, but there was a fairly severe reaction, with marked conjunctivitis. Two and one-half years afterward the patient returned complaining of a mist that had been gathering over the left eye for four months. An intumescent mature cataract of the left eye was found. There was no sign of metastasis.

HOLE AT THE MACULA. MR. F. A. WILLIAMSON-NOBLE.

This young woman noticed blurring in the right eye shortly before visiting the hospital two months ago. Vision, with correction, was less than 6/60, and there was a central scotoma for colors. In the macular region was a reddish area surrounded by a white penumbra, some yellowish-white dots and a few crystalline deposits. The left eye was normal. There was no history of injury. Two septic teeth were removed.

CONTACT GLASSES. MR. F. A. WILLIAMSON-NOBLE.

Since February 1932 this woman has been wearing contact glasses. She has 13 diopters of myopia and 4 diopters of myopic astigmatism in the right eye, and 10 diopters of myopia and 1 diopter of myopic astigmatism in the left eye. In the left fundus there is old chorioretinitis. The right eye, with correction, reads 6/12; the left reads 6/24; with a contact glass, both eyes read 6/12.

DISCUSSION

MR. RUGG-GUNN: These patients do get binocular vision with contact glasses. I have a patient, a taxi-driver, both of whose eyes were operated on. With contact glasses he gets binocular vision with full fields.

PITUITARY ADENOMA, RELIEVED BY OPERATION. MR. F. JULER.

This man, aged 39, attended Moorfields because of defective vision. For eleven months he had had severe left parietal headaches every day, and this was increased by eyestrain. Vision in the left eye had been defective for four weeks. For twelve months there had been a gradual loss of libido. Vision in the right eye was 6/6; that in the left, 6/24. The fields showed bitemporal hemianopia, complete in the left field, and incomplete below in the right field. The roentgenogram showed no abnormality of the sella turcica. At operation, a bluish suprasellar cyst was found and excised. Histologically, the tissue consisted of solid masses of cells of elongated shape and oval nuclei. It seemed to be an adenoma of the pars anterior or of the pars intermedia. Since the operation the fields have greatly improved, the right being almost full.

UNEXPLAINED BILATERAL PROPTOSIS. MR. E. F. KING.

This man, aged 50, was first seen as an outpatient, in August 1933, because he had had diplopia for four months. Since that date the condition has not materially changed. There are edema of the lower lids, proptosis, chemosis and congestion of conjunctival vessels. The right eye is convergent, with gross loss of lateral movement. Palpation gives an impression of solid edema, and the globes cannot be pushed back into the orbits. The pupils are equal and react to light and in accommodation. The tension is normal. There is sclerosis of the retinal vessels; otherwise, the fundi are normal. Vision in the right eye is 6/60, not improved; vision in the left eye is 6/36, not improved; the fields are full, and the blind spots normal.

DR. HILL (neurologist): The patient is free from signs of hyperthyroidism, and the only positive findings in regard to general disease were marked arteriosclerosis and considerable overcalcification in the skull; the diploe are fully calcified, and the choroid plexuses are visible on the roentgenogram. He shows no sign of venous back pressure in the eyes, and I think the edema in the orbits must be a lymphatic edema, probably a result of some lymphatic obstruction originating in the calcification. I think he might be treated by inserting threads subcutaneously between the edematous orbits and the normal subcutaneous tissue lower down in the face. His blood count, blood urea and blood sugar were normal.

Book Reviews

Lesions of the Fundus of the Eye Observed in Venezuela. By Dr. J. M. Espino. Work of Incorporation of the National Academy of Medicine of Venezuela. Paper. Pp. 181. Caracas: El Comercio, 1933.

This work comprises a statistical grouping of data based on the clinical histories and findings in cases seen in private practice in the city of Caracas during the period from March 1917 to November 1932. A total of 12,000 patients with ophthalmologic conditions is listed, in 9,450 of whom the fundus was examined. (The reason for the exclusion of the remaining 2,550 from this part of the routine is not stated.) Of those examined, 1,225 showed lesions in one or both fundi; among these were 153 patients who exhibited more than one classifiable condition.

The lesions are grouped in a purely descriptive or topographic manner. Regarding the general condition of the patients, the author has limited himself to a notation of the disease or diseases present at the time of observation of the pathologic changes in the fundus. Frequently no speculation is made as to the cause and effect, this being left to the judgment of the reader. There are no illustrations. Because of the large number of cases studied, only such notes as embrace the more important details of the interesting and notable lesions are appended.

Optic neuritis	44
Optic atrophy	106
Retinitis	122
Detachment of the retina	27
Neuro-epithelioma of the retina	1
Neurorentinitis: neurochorioretinitis	135
Choroiditis; chorioretinitis	339
Posterior sclerochoroiditis; myopic choroiditis; myopic fundus	161
Tumor of the choroid (leukosarcoma)	1
Glaucomatous fundus	135
Retinal angiosclerosis	156
Circulatory and vascular alterations	114
Traumatic lesions	15
Congenital conditions	22

The bibliography consists simply of the names of the authors quoted, without note of the publications.

The volume shows the result of an enormous amount of painstaking work; and its perusal is worth while because of the great quantity of clinical material included. If at times the reader's method of classification differs from that of the author, this makes the book in no way less valuable.

SOLON L. RHODE.

Directory of Ophthalmologic Societies *

INTERNATIONAL

International Association for Prevention of Blindness

President: Prof. F. de Lapersonne, 217 Faubourg St., Honoré, Paris.
Secretary-General pro tem: Dr. A. Churchill, 66 Boulevard St. Michel, Paris.

International Ophthalmologic Congress

Secretary: Dr. Francisco Poyales, Olozaga 3, Madrid.

FOREIGN

British Medical Association, Section on Ophthalmology

President: Dr. Frank H. Crawley, 5 Fitzwilliam Pl., Dublin.

Ophthalmology Society of Bombay

President: Dr. D. D. Sathaye, 127 Girgaum Road, Bombay 4.
Secretary: Dr. H. D. Dastur, Dadar, Bombay 14.
Place: H. B. A. Free Ophthalmic Hospital, Parel, Bombay 12. Time: First Friday of every month.

Ophthalmological Society of the United Kingdom

President: Mr. A. H. H. Sinclair, 6 Charlotte Square, Edinburgh.
Secretary: Mr. Humphrey Neame, 56 Portland Place, London, W. I.

Oxford Ophthalmological Congress

Master: Dr. Cyril Walker, Bristol, England.
Secretary: Dr. C. G. Russ Wood, Hill House, Abberbury Rd., Iffley, Oxford, England.

Royal Society of Medicine, Section of Ophthalmology

President: Dr. A. C. Hudson, 50, Queen Anne St., W. 1, London.
Secretary: Dr. J. D. Magor Carbell, 27, Weymouth St., W. 1, London.

Société Française d'Ophthalmologie

Secretary: Dr. René Onfray, 6 avenue de la Motte Picquet, Paris 7è.

Society of Swedish Ophthalmologists

President: Prof. F. Berg, Uppsala, Sweden.
Secretary: Dr. K. O. Granström, Södermalmstorg 4 III tr., Stockholm, Sö., Sweden.

German Ophthalmological Society

Secretary: Prof. A. Wagenmann, Heidelberg.

NATIONAL

American Medical Association, Scientific Assembly, Section on Ophthalmology

Chairman: Dr. Arthur J. Bedell, 344 State St., Albany, N. Y.
Secretary: Dr. Parker Heath, 1553 Woodward Ave., Detroit.
Place: Atlantic City, N. J. Time: June 10-14, 1935.

* Secretaries of societies are requested to furnish the information necessary to make this list complete and to keep it up to date.

AMERICAN ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. J. M. Wheeler, 635 W. 165th St., New York.
President-Elect: Dr. Wells P. Eagleton, 15 Lombardy St., Newark, N. J.
Executive Secretary-Treasurer: Dr. William P. Wherry, 1500 Medical Arts Bldg., Omaha.
Place: Chicago. Time: Sept. 9-14, 1934.

AMERICAN OPHTHALMOLOGICAL SOCIETY

President: Dr. W. G. M. Byers, 1458 Mountain St., Montreal.
Secretary-Treasurer: Dr. J. Milton Griscom, 2213 Walnut St., Philadelphia.

NATIONAL SOCIETY FOR THE PREVENTION OF BLINDNESS

President: Mr. William Fellows Morgan.
Managing Director: Mr. Lewis H. Carris, 50 W. 50th St., New York.
Place: New York. Time: November, 1934.

SECTIONAL

NEW ENGLAND OPHTHALMOLOGICAL SOCIETY

President: Dr. Hugo B. C. Riemer, 128 Newbury St., Boston.
Secretary-Treasurer: Dr. Benjamin Sachs, 483 Beacon St., Boston.
Place: Massachusetts Eye and Ear Infirmary, 243 Charles St., Boston. Time: 8 p. m., third Tuesday of each month from November to April, inclusive.

PACIFIC COAST OTO-OPHTHALMOLOGICAL SOCIETY

President: Dr. A. W. Morse, 66 W. Park St., Butte, Mont.
Secretary-Treasurer: Dr. Frederick C. Cordes, 384 Post St., San Francisco.

PUGET SOUND ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. Francis A. Brugman, 509 Olive St., Seattle.
Secretary-Treasurer: Dr. Noble Dick, 1115 Terry Ave., Seattle.
Time: 8 p. m., third Tuesday of each month, except July and August.

ROCK RIVER VALLEY EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. Harry Shearer, 405 E. Grand Ave., Beloit, Wis.
Secretary-Treasurer: Dr. W. S. Beyer, 1101 Talcott Bldg., Rockford, Ill.
Place: Rockford, Ill., Janesville or Beloit, Wis. Time: Third Tuesday of each month.

SAGINAW VALLEY ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. A. R. McKinney, 703 Second National Bank Bldg., Saginaw, Mich.
Secretary-Treasurer: Dr. W. K. Slack, 308 Eddy Bldg., Saginaw, Mich.
Place: Saginaw, Mich., or Bay City, Mich. Time: First Friday of each month, except July and August.

SIOUX VALLEY EYE AND EAR ACADEMY

President: Dr. John B. Gregg, 101 S. Main St., Sioux Falls, S. Dakota.
Secretary-Treasurer: Dr. F. H. Roost, 420 Trimble Bldg., Sioux City, Iowa.

SOUTHERN MEDICAL ASSOCIATION, SECTION ON EYE, EAR, NOSE AND THROAT

Chairman: Dr. Dunbar Roy, 113 N. Pryor St., Atlanta, Ga.
Secretary: Dr. William A. Wagner, 914 American Bank Bldg., New Orleans.
Place: New Orleans. Time: 1934.

SOUTHWESTERN MICHIGAN TRIOLOGICAL SOCIETY

President: Dr. Carl Snapp, 604 Medical Arts Bldg., Grand Rapids.
Secretary-Treasurer: Dr. Lee O. Grant, 420 Medical Arts Bldg., Grand Rapids.
Time: Third Thursday of alternate months.

WESTERN PENNSYLVANIA EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. O. G. A. Barker, 532 Main St., Johnstown.
Secretary-Treasurer: Dr. C. W. Beals, Weber Bldg., DuBois.
Time: Third Thursday of October and May.

STATE

COLORADO OPHTHALMOLOGICAL SOCIETY

President: A presiding officer is selected for each meeting alternately until all members have served.
Secretary: Dr. Edna M. Reynolds, 920 Republic Bldg., Denver.
Place: Capitol Life Building, Denver. Time: 7:30 p. m., third Saturday of the month, October to April, inclusive.

CONNECTICUT STATE MEDICAL SOCIETY, SECTION ON EYE, EAR, NOSE AND THROAT

President: Dr. Edward J. Whalen, 750 Main St., Hartford.
Secretary-Treasurer: Dr. Walter L. Hogan, 750 Main St., Hartford.
Time: May, November.

EYE, EAR, NOSE AND THROAT CLUB OF GEORGIA

President: Dr. W. R. Bedingfield, 753 Broad St., Augusta.
Secretary-Treasurer: Dr. J. A. Smith, 700 Spring St., Macon.

INDIANA ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. J. Kent Leasure, 23 E. Ohio St., Indianapolis.
Secretary: Dr. Fred Overman, 611 Hume-Mansure Bldg., Indianapolis.
Place: Richmond. Time: Dec. 12, 1934.

IOWA ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. W. F. Boiler, 105 E. Iowa Ave., Iowa City.
Secretary-Treasurer: Dr. Lawrence A. Taylor, 118 E. Second St., Ottumwa.

MICHIGAN STATE MEDICAL SOCIETY, SECTION OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

Chairman: Dr. Peter T. Grant, 148 Monroe Ave., Grand Rapids.
Secretary: Dr. Ralph B. Fast, 1410 Bank of Kalamazoo Bldg., Kalamazoo.
Place: Battle Creek. Time: 1934.

MINNESOTA ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Gordon B. New, Mayo Clinic, Rochester.
Secretary-Treasurer: Dr. Walter E. Camp, Medical Arts Bldg., Minneapolis.
Time: Second Friday of each month from October to May.

MONTANA ACADEMY OF OTO-OPHTHALMOLOGY

President: Dr. Lawrence G. Dunlap, 101 Main St., Anaconda.
Secretary: Dr. A. W. Morse, Phoenix Bldg., Butte.

NEW JERSEY STATE MEDICAL SOCIETY, SECTION ON OPHTHALMOLOGY, OTOLOGY AND RHINOLARYNGOLOGY

Chairman: Dr. S. T. Hubbard, 294 State St., Hackensack.
Secretary: Dr. G. S. Laird, 125 Central Ave., Westfield.

NEW YORK STATE MEDICAL SOCIETY, EYE, EAR, NOSE AND THROAT SECTION

Chairman: Dr. Frank M. Sulzman, 1831 Fifth Ave., Troy.
Secretary: Dr. Webb W. Weeks, 20 E. 53rd St., New York.

NORTH DAKOTA ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. H. O. Ruud, 318 De Mers Ave., Grand Forks.
Secretary-Treasurer: Dr. F. L. Wicks, 516 Fourth St., Valley City.
Place: Valley City. Time: 1934.

OREGON ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. A. B. Dykman, Medical Dental Bldg., Portland.
Secretary-Treasurer: Dr. Andrew J. Browning, 418 Mayer Bldg., Portland.
Place: Good Samaritan Hospital. Time: Third Tuesday of each month.

RHODE ISLAND OPHTHALMOLOGICAL AND OTOLOGICAL SOCIETY

President: Dr. Robert C. O'Neil, 105 Waterman St., Providence.
Secretary and Treasurer: Dr. Gordon J. McCurdy, 122 Waterman St., Providence.
Place: Rhode Island Medical Library. Time: 8:30 p. m., second Thursday in October, December, February and April.

SOUTH CAROLINA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. Walter Bristow, Medical Bldg., Columbia.
Secretary: Dr. J. W. Jervey, Jr., 101 Church St., Greenville.

TENNESSEE ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. C. D. Blassingame, 899 Madison Ave., Memphis.
Secretary-Treasurer: Dr. Ralph O. Rychener, 130 Madison Ave., Memphis.

TEXAS OPHTHALMOLOGICAL AND OTO-LARYNGOLOGICAL SOCIETY

President: Dr. C. C. Cody, 1304 Walker Ave., Houston.
Secretary: Dr. O. M. Marchman, 1719 Pacific Ave., Dallas.

UTAH OPHTHALMOLOGICAL SOCIETY

President: Dr. F. R. Slopanskey, 9 Exchange Pl., Salt Lake City.
Secretary-Treasurer: Dr. T. F. Welsh, 920 Boston Bldg., Salt Lake City.
Time: Third Monday of each month.

VIRGINIA SOCIETY OF OTO-LARYNGOLOGY AND OPHTHALMOLOGY

President: Dr. Beverley R. Kennon, 142 W. York St., Norfolk.
Secretary-Treasurer: Dr. George G. Hankins, Medical Arts Bldg., Newport News.

WEST VIRGINIA STATE MEDICAL ASSOCIATION, EYE, EAR, NOSE AND THROAT SECTION

President: Dr. Harry V. Thomas, 95 Fairmont Ave., Fairmont.
Secretary: Dr. W. F. Beckner, 1050 Fifth Ave., Huntington.

LOCAL

ACADEMY OF MEDICINE OF NORTHERN NEW JERSEY, SECTION ON EYE, EAR, NOSE AND THROAT

Chairman: Dr. Browne Morgan, 260 Liberty St., Bloomfield.
Secretary: Dr. A. Russell Sherman, 671 Broad St., Newark.
Place: 91 Lincoln Park South, Newark. Time: 8:45 p. m., second Monday of each month.

ACADEMY OF MEDICINE OF TORONTO, SECTION OF OPHTHALMOLOGY

Chairman: Dr. Colin Campbell, 170 St. George St., Toronto.
Secretary: Dr. H. M. Macrae, 170 St. George St., Toronto.
Time: First Monday of winter months.

ATLANTA EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. W. L. McDougall, 50 Whitehall St., Atlanta, Ga.
Secretary: Dr. William O. Martin, Jr., 384 Peachtree St., Atlanta, Ga.
Place: Academy of Medicine, 38 Prescott St., N. E. Time: 8 p. m., last Thursday of each month.

Baltimore Medical Society, Section on Ophthalmology

Chairman: Dr. Lloyd B. Whitham, Mt. Vernon Pl. and Charles St., Baltimore.
Secretary: Dr. Mary L. Small, 18 W. Read St., Baltimore.
Place: Medical and Chirurgical Faculty, 1211 Cathedral St. Time: 8:30 p. m., fourth Thursday of each month from October to May.

Brooklyn Ophthalmological Society

President: Dr. John H. Bailey, 855 St. Marks Ave., Brooklyn.
Secretary-Treasurer: Dr. George Freiman, 703 Grand St., Brooklyn.
Place: Kings County Medical Society Bldg., 1313 Bedford Ave. Time: Third Thursday in February, April, May, October and December.

Buffalo Ophthalmologic Club

President: Dr. H. H. Glosser, 448 Franklin St., Buffalo.
Secretary-Treasurer: Dr. I. J. Koenig, 40 North St., Buffalo.

Chicago Ophthalmological Society

President: Dr. Dwight C. Orcutt, 30 N. Michigan Ave., Chicago.
Secretary: Dr. Richard C. Gamble, 30 N. Michigan Ave., Chicago.
Place: Medinah Michigan Avenue Club, 505 N. Michigan Ave. Time: Third Monday of each month from October to May.

Cleveland Academy of Medicine, Section of Ophthalmology and Otolaryngology

Chairman: Dr. A. L. Stotter, 1148 Euclid Ave., Cleveland.
Secretary: Dr. James T. Collins, 1105 Medical Arts Bldg., Cleveland.
Place: Winton Hotel. Time: Fourth Friday of each month.

Cleveland Ophthalmological Club

Chairman: Dr. W. P. Chamberlain, 7405 Detroit Ave., Cleveland.
Secretary: Dr. A. D. Ruedemann, Cleveland Clinic, Cleveland.

College of Physicians, Philadelphia, Section on Ophthalmology

Chairman: Dr. John Milton Griscom, 2213 Walnut St., Philadelphia.
Clerk: Dr. Alexander G. Fewell, 1924 Pine St., Philadelphia.
Time: Third Thursday of every month from October to April, inclusive.

Columbus Ophthalmological and Oto-Laryngological Society

Chairman: Dr. Albert D. Frost, 681 E. Broad St., Columbus, Ohio.
Secretary-Treasurer: Dr. M. Goldberg, 328 E. State St., Columbus, Ohio.
Time: First Monday of each month.

Dallas Academy of Ophthalmology and Oto-Laryngology

President: Dr. T. S. Love, 1719 Pacific Ave., Dallas, Texas.
Secretary: Dr. Daniel Brannin, 1719 Pacific Ave., Dallas, Texas.
Place: Dallas Athletic Club. Time: 6:30 p. m., first Tuesday of each month from October to June. The November, January and March meetings are devoted to clinical work.

Des Moines Academy of Ophthalmology and Otolaryngology

President: Dr. C. C. Walker, 410 6th Ave., Des Moines, Iowa.
Secretary-Treasurer: Dr. Grace Doane, 614 Bankers Trust Bldg., Des Moines, Iowa.
Time: 7:45 p. m., third Monday of every month, September to May.

Detroit Ophthalmological Club

Chairman: Members rotate alphabetically.
Secretary: Dr. Raymond S. Goux, David Whitney Bldg., Detroit.
Time: 6:30 p. m., first Wednesday of each month.

Eastern New York Eye, Ear, Nose and Throat Association

President: Dr. Edward B. O'Keefe, 901 State St., Schenectady.
Secretary-Treasurer: Dr. W. C. Mott, 118 Washington Ave., Albany.
Time: Third Wednesday in October, November, March, April, May and June.

Fort Worth Eye, Ear, Nose and Throat Society

President: Dr. E. L. Howard, 600 W. 10th St., Fort Worth, Texas.
Secretary-Treasurer: Dr. Van D. Rathgeber, 1012 Medical Arts Bldg., Fort Worth.
Place: Medical Hall, Medical Arts Bldg. Time: 7:30 p. m., first Friday of each month except July and August.

Grand Rapids Eye, Ear, Nose and Throat Society

President: Dr. Carl F. Snapp, 26 Sheldon Ave., Grand Rapids, Mich.
Secretary-Treasurer: Dr. Walter W. Oliver, 320 Medical Arts Bldg., Grand Rapids, Mich.
Place: Various local hospitals. Time: Third Thursday of alternating months, September to May.

Houston Academy of Medicine, Eye, Ear, Nose and Throat Section

President: Dr. E. L. Goar, 1304 Walker Ave., Houston, Texas.
Secretary: Dr. J. C. Dickson, 622 Medical Arts Bldg., Houston, Texas.
Place: Medical Arts Bldg., Harris County Medical Society Rooms. Time: 8 p. m., second Thursday of each month from September to June.

Indianapolis Ophthalmological and Otolaryngological Society

President: Dr. D. A. Bartley, 23 E. Ohio St., Indianapolis.
Secretary: Dr. Kenneth L. Craft, 23 E. Ohio St., Indianapolis.
Place: University Club. Time: 6:30 p. m., second Thursday of each month from October to May.

Kansas City Society of Ophthalmology and Oto-Laryngology

President: Dr. M. B. Simpson, 1103 Grand Ave., Kansas City, Mo.
Secretary: Dr. Harold B. Hedrick, 618 Professional Bldg., Kansas City, Mo.
Time: Third Thursday of each month from September to May. The November, January and March meetings are devoted to clinical work.

Long Beach Eye, Ear, Nose and Throat Society

Chairman: Dr. Russell T. Uhls, 110 Pine Ave., Long Beach, Calif.
Secretary-Treasurer: Dr. Kenneth C. Brandenburg, 707 Security Bldg., Long Beach, Calif.
Place: Professional Bldg. Time: Last Wednesday of each month from October to May.

Los Angeles Society of Ophthalmology and Oto-Laryngology

President: Dr. F. H. Brandt, 1136 W. Sixth St., Los Angeles.
Secretary-Treasurer: Dr. Pierre Viole, 1930 Wilshire Blvd., Los Angeles.
Place: Los Angeles County Medical Association Bldg., 1925 Wilshire Blvd. Time: 6:30 p. m., fourth Monday of each month from September to May, inclusive.

Louisville Eye, Ear, Nose and Throat Society

President: Dr. Charles Dwight Townes, 332 W. Broadway, Louisville, Ky.
Secretary-Treasurer: Dr. J. S. Bumgardner, 1103 Heyburn Bldg., Louisville, Ky.
Place: French Village. Time: Second Thursday of each month, September to May, inclusive, at 6:30 p. m.

Medical Society of the District of Columbia, Section of Ophthalmology and Oto-Laryngology

Chairman: Dr. George B. Trible, 1801 I St., Washington.
Secretary: Dr. Harry F. Davies, 1835 I St., Washington.
Place: 1718 M St., N. W. Time: 8 p. m., third Friday of each month from October to April, inclusive.

Memphis Society of Ophthalmology and Otolaryngology

Chairman: Each member, in alphabetical order.
Secretary: Dr. R. O. Rychener, Exchange Bldg., Memphis, Tenn.
Place: Eye Clinic of Memphis Eye, Ear, Nose and Throat Hospital. Time: 8 p. m., second Tuesday of each month.

Milwaukee Oto-Ophthalmic Society

Secretary-Treasurer: Dr. Herbert G. Schmidt, 238 W. Wisconsin Ave., Milwaukee.
President: Dr. O. P. Schoofs, 324 E. Wisconsin Ave., Milwaukee.
Place: University Club. Time: 6:30 p. m., fourth Tuesday of each month.

Montreal Ophthalmological Society

President: Dr. J. A. MacMillan, 1410 Stanley St., Montreal, Canada.
Secretary: Dr. A. L. Guertin, 410 Sherbrooke St., West, Montreal, Canada.
Time: Second Thursday of October, December, February and April.

Nashville Academy of Ophthalmology and Otolaryngology

Chairman: Dr. Frederick E. Hasty, 119, 7th Ave. N., Nashville, Tenn.
Secretary-Treasurer: Dr. Kate Savage Zerfoss, 803 Medical Arts Bldg., Nashville, Tenn.
Place: St. Thomas Hospital. Time: 8 p. m., third Monday of each month from October to June.

New Orleans Ophthalmological and Otolaryngological Society

President: Dr. M. F. Meyer, Canal Bank Bldg., New Orleans.
Secretary-Treasurer: Dr. R. H. Fisher, Chess and Checker Club, New Orleans.
Place: Eye, Ear, Nose and Throat Hospital. Time: Third Thursday of each month from October to June.

New York Academy of Medicine, Section of Ophthalmology

Chairman: Dr. Webb William Weeks, 20 E. 53rd St., New York.
Secretary: Dr. Guernsey Frey, 121 E. 60th St., New York.
Time: 8:30 p. m., third Monday of every month from October to May, inclusive.

Omaha and Council Bluffs Ophthalmological and Oto-Laryngological Society

President: Dr. Abbott M. Dean, 532, 1st Ave., Council Bluffs, Iowa.
Secretary-Treasurer: Dr. Robert H. Rasgorshek, 425 Aquila Court, Omaha.
Place: Omaha Club, 20th and Douglas Sts., Omaha. Time: 6 p. m., dinner; 7 p. m., program; third Wednesday of each month from October to May.

Philadelphia County Medical Society, Eye Section

Chairman: Dr. Charles R. Heed, 1205 Spruce St., Philadelphia.
Secretary: Dr. Sidney L. Olsho, 235 S. 15th St., Philadelphia.
Time: Second Tuesday of each month from October to May.

Pittsburgh Ophthalmological Society

President: Dr. Edward B. Heckel, Jenkins Arcade Bldg., Pittsburgh.
Secretary: Dr. George H. Shuman, Park Bldg., Pittsburgh.
Place: Pittsburgh Academy of Medicine Bldg. Time: Fourth Monday of each month, except June, July, August and September.

Pittsburgh Slit-Lamp Society

President: Dr. W. W. Blair, 121 University Pl., Pittsburgh.
Secretary: Dr. George H. Shuman, Park Bldg., Pittsburgh.
Place: 121 University Pl. Time: Second Monday evening of every month, except June, July, August and September.

Richmond Ophthalmological and Oto-Laryngological Society

President: Dr. F. H. Lee, 501 E. Franklin St., Richmond, Va.
Secretary: Dr. William R. Weisiger, Medical Arts Bldg., Richmond, Va.
Place: Westmoreland Club. Time: 6 p. m., second Monday of each month from October to May.

Rochester Eye, Ear, Nose and Throat Society

President: Dr. E. W. O'Brien, 5 N. Goodman St., Rochester, N. Y.
Secretary-Treasurer: Dr. W. D. Edwards, 389 Monroe Ave., Rochester, N. Y.
Place: Rochester Medical Association, 113 Prince St. Time: 8 p. m., third Monday of each month from October to May.

St. Louis Ophthalmic Society

President: Dr. E. C. Spitze, 234 Collinsville Ave., East St. Louis, Ill.
Secretary: Dr. Carl T. Eber, 1006 Carleton Bldg., St. Louis.
Place: Oscar Johnson Institute. Time: Clinical meeting 5:30 p. m., dinner and scientific meeting 6:30 p. m., fourth Friday of each month from October to April, inclusive, except December.

San Antonio Ophthalmolo-Oto-Laryngological Society

President: Dr. Robert E. Parrish, 712 Medical Arts Bldg., San Antonio, Texas.
Secretary-Treasurer: Dr. Frank Steed, Nix Professional Bldg., San Antonio, Texas.
Place: Bexar County Medical Library. Time: 8 p. m., first Tuesday of each month from October to May.

San Francisco County Medical Society, Section on Eye, Ear, Nose and Throat

Chairman: Dr. Robert S. Irvine, 490 Post St., San Francisco.
Secretary: Dr. Frank H. Rodin, 490 Post St., San Francisco.
Place: Society's Building, 2180 Washington St., San Francisco.
Time: Fourth Tuesday of every month except May, June, July and December.

Shreveport Eye, Ear, Nose and Throat Society

President: Dr. W. L. Atkins, 940 Margaret Pl., Shreveport, La.
Secretary-Treasurer: Dr. Leon F. Gray, 1214 Slattery Bldg., Shreveport, La.
Place: 1240 Texas Ave. Time: 8:15 p. m., fourth Tuesday of every month except June, July and December.

Spokane Academy of Ophthalmology and Oto-Laryngology

President: Dr. Marc Anthony, 422 Riverside Ave., Spokane, Wash.
Secretary: Dr. Raymond A. Lower, 1455 Paulsen Medical and Dental Bldg., Spokane, Wash.
Place: Paulsen Medical and Dental Library. Time: 8 p. m., fourth Tuesday of each month except June, July and August.

Syracuse Eye, Ear, Nose and Throat Society

President: Dr. H. O. Brust, 820 S. Crouse Ave., Syracuse, N. Y.
Secretary-Treasurer: Dr. W. J. Werfelmann, 109 S. Warren St., Syracuse, N. Y.
Place: University Club. Time: Second Friday of each month except June, July and August.